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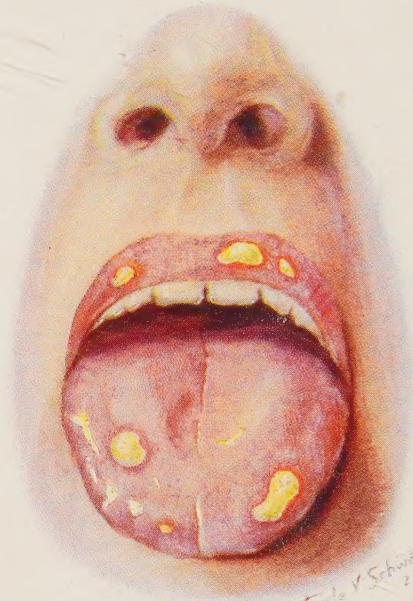
**DISEASES OF  
THE DIGESTIVE SYSTEM**



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FRONTISPIECE (FIG. 6.).—LESIONS ON LIPS AND TONGUE  
IN HERPETIC STOMATITIS.



CLINICAL PEDIATRICS

# DISEASES OF THE DIGESTIVE SYSTEM OF CHILDHOOD

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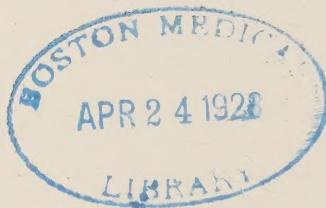


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## PREFACE

This volume is devoted to a consideration of the digestive tract during the early periods of life, both in health and in disease. It is intended for the physician in general practice who feels the need of learning all that he can about the peculiarities of children, the disabilities to which they are liable, and the most satisfactory methods of diagnosis and relief.

The subject of the digestive system is not so narrow as it would seem at first thought. True, the stomach in childhood offers little or practical importance from a clinical standpoint. Fortunately, the diseases of the intestinal tract are becoming rarer, but many affections of other regions act both directly and indirectly to disturb the digestive system. Also the diet, nutrition, environment and various habits may, any or all of them, influence this system.

No greater mistake can be made than to confine one's efforts to the relief of such common symptoms as vomiting, diarrhea or constipation, and neglect the search for the cause.

A complete physical examination is to be emphasized as a necessity in all cases. As a common illustration of this fact, one has only to recall the symptoms simulating appendicitis which are frequently found in lobar pneumonia.

Throughout this volume will be found references to the onset and presence of many infections peculiar to childhood such as the exanthemata, inflammations of the ear, nose and throat and many other diseases in which loss of appetite and gastro-intestinal disturbances are common and secondary to the parenteral disease.

The practitioner therefore must not isolate—either in his diagnosis or treatment—the primary digestive tract from a broad consideration of the child as a whole.

FRANK C. NEFF.





## PUBLISHERS' ANNOUNCEMENT

The publishers take pleasure in presenting to the medical profession the series of monographs of which this volume forms a unit.

The many inquiries which reached them proved, in advance of publication, that the work should be in monographic form and clinical in its presentation.

The series when completed will, they believe, be the most useful for the audience for whom it is written, the general practitioner of medicine, that has been presented in its particular field.

The authors are all men of wide experience and, in the main, teachers. The combination makes the work authoritative and of the utmost service in a field which has been often termed a "therapeutic specialty."



# CONTENTS

CHAPTER	PAGE
I. THE MOUTH . . . . .	I
Characteristics at Birth . . . . .	I
Mechanism of Sucking . . . . .	2
Microorganisms . . . . .	2
Malformations of the Structure of the Mouth . . . . .	2
Tongue-Tie . . . . .	2
Tongue-Swallowing . . . . .	4
Tumors and Deformities of the Tongue . . . . .	4
Cyst . . . . .	6
Harelip and Cleft-Palate . . . . .	6
II. DISEASES OF THE MOUTH . . . . .	12
The Lip . . . . .	12
Herpes of the Lip . . . . .	12
The Frenum . . . . .	12
Simple Ulcer . . . . .	12
Riga's Disease . . . . .	12
The Gums . . . . .	13
Gingivitis . . . . .	13
Abscess and Periostitis . . . . .	13
Hypertrophy . . . . .	14
Scurvy . . . . .	14
The Tongue . . . . .	15
Geographic Tongue . . . . .	15
Glossitis . . . . .	16
The Jaw . . . . .	16
Necrosis . . . . .	16
Tumor . . . . .	16
Sarcoma . . . . .	17
Syphilis of the Mouth . . . . .	17
III. DISEASES OF THE MOUTH ( <i>Continued</i> ) . . . . .	19
Stomatitis . . . . .	19
General Consideration . . . . .	19
Etiology . . . . .	19
Thrush . . . . .	19
Etiology . . . . .	20
Lesions . . . . .	20
Symptoms . . . . .	21
Diagnosis . . . . .	21
Treatment . . . . .	22
Prognosis . . . . .	22
Catarrhal Stomatitis . . . . .	22



CHAPTER	PAGE
Symptoms . . . . .	23
Treatment . . . . .	23
Herpetic Stomatitis (Maculofibrinous or Follicular Stomatitis)	24
Etiology . . . . .	24
Symptoms . . . . .	25
Treatment . . . . .	26
Ulcerative Stomatitis . . . . .	26
Nomenclature . . . . .	27
Etiology . . . . .	27
Lesions . . . . .	27
Symptoms . . . . .	28
Diagnosis . . . . .	29
Treatment . . . . .	29
Gangrenous Stomatitis (Cancrum Oris, Noma) . . . . .	30
Etiology . . . . .	31
Lesions . . . . .	32
Symptoms . . . . .	32
Diagnosis . . . . .	33
Prognosis . . . . .	33
Treatment . . . . .	34
Prophylaxis . . . . .	35
Ulceration of the Hard Palate . . . . .	35
Mercurial Stomatitis . . . . .	36
IV. AFFECTIONS OF THE PHARYNX AND TONSILS . . . . .	37
The Pharynx . . . . .	37
Anatomical Characteristics . . . . .	37
Foreign Bodies in the Pharynx . . . . .	37
Disturbances of the Uvula . . . . .	38
Treatment . . . . .	38
Acute Pharyngitis . . . . .	39
Actinomycosis . . . . .	40
The Tonsils (Acute Inflammation of the Tonsils) . . . . .	40
Tonsillar Diphtheria . . . . .	40
Scarlatinal Tonsillitis . . . . .	41
Vincent's Angina . . . . .	41
Peritonsillar Abscess (Quinsy) . . . . .	42
Retropharyngeal Abscess (Retropharyngeal Lymphadenitis)	43
Acute Follicular Tonsillitis . . . . .	46
The Rôle of Lymphoid Diseases in the Pharynx and Fauces .	48
Anatomical Changes in the Tonsil . . . . .	48
Adenoid Vegetations . . . . .	50
V. THE SALIVARY GLANDS . . . . .	52
Anatomical and Functional Consideration . . . . .	52
Disturbances of Salivary Secretion . . . . .	53
Abnormal Development . . . . .	53
Salivary Cyst . . . . .	54
Mixed Tumors of the Parotid . . . . .	54
Mikulicz's Disease . . . . .	55

# CONTENTS

xi

## CHAPTER

PAGE

Epidemic Parotitis . . . . .	56
Parotitis (Non-epidemic) . . . . .	56
Submaxillary Inflammation . . . . .	58
Sublingual Inflammation . . . . .	58
Salivary Stones . . . . .	58
Syphilis . . . . .	59
Lymphatic Enlargement . . . . .	59

## VI. THE TEETH . . . . . 60

Dentition . . . . .	60
The Deciduous Teeth . . . . .	60
Eruption . . . . .	60
Loss of Teeth . . . . .	61
Disturbances during Eruption of the Teeth . . . . .	62
Abnormalities . . . . .	63
Diseases of the Temporary Teeth . . . . .	63
The Permanent Teeth in Childhood . . . . .	65
Abnormalities . . . . .	65
Caries . . . . .	66
Trauma . . . . .	68
Malocclusion . . . . .	68
Syphilis of the Permanent Teeth . . . . .	69
Dental Hygiene . . . . .	69
Prenatal Care . . . . .	70
The Preschool Child . . . . .	70

## VII. THE ESOPHAGUS . . . . . 72

Congenital Malformation . . . . .	72
Congenital Narrowing . . . . .	74
Diseases of the Esophagus . . . . .	74
Foreign Bodies in the Esophagus . . . . .	76
Lodgment of Safety Pin . . . . .	76
Spasm of the Esophagus . . . . .	80
Stricture of Esophagus . . . . .	80

## VIII. THE STOMACH AND ITS DISTURBANCES . . . . . 86

Anatomy and Physiology . . . . .	86
Physiology and Gastric Digestion . . . . .	89
Vomiting in Infancy . . . . .	92
Simple Regurgitation . . . . .	92
Mechanical Vomiting . . . . .	92
Causes Inherent in the Food . . . . .	93
Gastric Insufficiency (Atony of the Stomach) . . . . .	94
Eruption of Teeth . . . . .	94
Toxic Vomiting . . . . .	95
So-called Intestinal Influenza . . . . .	95
Vomiting of Central Origin . . . . .	95
Influence of Previously Frozen Milk . . . . .	95
Treatment of Vomiting . . . . .	96

CHAPTER	PAGE
IX. NURSING AND ALIMENTARY DISTURBANCES IN INFANCY . . .	97
Nursing Difficulties . . . . .	97
Colic in Infancy . . . . .	98
Digestive Disturbances in Early Infancy . . . . .	100
Starvation Fever in the Newly Born . . . . .	101
Heat Exhaustion in Infancy . . . . .	102
Alimentary Anemia in Infancy . . . . .	102
X. DISEASES OF THE STOMACH . . . . .	104
Acute Gastritis . . . . .	104
Catarrhal Gastritis . . . . .	104
Symptoms . . . . .	105
Treatment . . . . .	105
Acute Toxic Gastritis . . . . .	106
Pathological Lesions . . . . .	106
Symptoms . . . . .	106
Treatment . . . . .	106
Chronic Gastric Indigestion . . . . .	107
Treatment . . . . .	107
Dilatation of the Stomach . . . . .	108
Hemorrhage . . . . .	108
Ulcer of the Stomach . . . . .	109
Occurrence . . . . .	109
Causes . . . . .	110
Pathological Lesions . . . . .	110
Symptoms . . . . .	111
Foreign Bodies in the Stomach . . . . .	111
Treatment . . . . .	112
Malformations and Tumors . . . . .	113
Hypertrophic Pyloric Stenosis . . . . .	113
Descriptive Summary . . . . .	113
Nomenclature and History . . . . .	113
Etiology . . . . .	114
Incidence . . . . .	114
Physiology and Pathology . . . . .	115
Symptoms . . . . .	115
Diagnosis and Differentiation . . . . .	119
Treatment . . . . .	121
Stomach Washing . . . . .	121
Dietary . . . . .	121
Medical Treatment . . . . .	121
Surgical Treatment . . . . .	123
Prognosis . . . . .	124
Pyloric Stenosis in Older Children . . . . .	125
Gastro-Enterospasm of Infancy (Pylorospasm) . . . . .	126
Clinical Description . . . . .	126
Etiology . . . . .	126
Physiology and Pathology . . . . .	127
Symptoms . . . . .	127
Treatment . . . . .	127

# CONTENTS

xiii

## CHAPTER

PAGE

Diagnosis and Differentiation . . . . .	128
Prognosis . . . . .	129

## XI. RUMINATION AND CYCLIC VOMITING . . . . . 130

Rumination . . . . .	130
Descriptive Summary . . . . .	130
Nomenclature . . . . .	130
Etiology . . . . .	130
Mechanism . . . . .	130
Symptoms . . . . .	131
Course . . . . .	132
Diagnosis . . . . .	132
Treatment . . . . .	132
In Older Children . . . . .	133
Cyclic Vomiting . . . . .	134
Descriptive Summary . . . . .	134
Incidence . . . . .	134
Etiology . . . . .	134
Symptoms . . . . .	135
Diagnosis . . . . .	136
Prognosis . . . . .	136
Treatment . . . . .	137

## XII. THE INTESTINES . . . . . 139

Anatomy and Physiology . . . . .	139
Physiology of the Duodenum . . . . .	140
Intestinal Digestion . . . . .	141
Stools in Infancy . . . . .	142
Bacteria of the Infant Intestinal Tract . . . . .	144
Peptic Ulcers of the Duodenum . . . . .	145

## XIII. THE INTESTINES (*Continued*) . . . . . 149

Diarrhea . . . . .	149
General Consideration . . . . .	149
Dyspepsia in Premature Infants . . . . .	153
Treatment . . . . .	156
Dyspepsia of the Breast-fed Infant . . . . .	158
Etiology . . . . .	158
Symptoms . . . . .	159
Diagnosis . . . . .	160
Treatment . . . . .	160
Prognosis . . . . .	161
Dyspepsia of the Artificially Fed Infant . . . . .	161
Etiology . . . . .	161
Physiological Chemistry . . . . .	162
Pathological Changes . . . . .	162
Symptoms . . . . .	162
Treatment . . . . .	163
Digestive Injury from Starch . . . . .	164
Symptoms . . . . .	164
Treatment . . . . .	165



CHAPTER	PAGE
XIV. THE INTESTINES ( <i>Continued</i> ) . . . . .	166
Diarrhea ( <i>Continued</i> ) . . . . .	166
Acute Intestinal Indigestion . . . . .	166
Symptoms and Course . . . . .	168
Diagnosis . . . . .	169
Complication . . . . .	170
Prevention . . . . .	170
Treatment . . . . .	170
XV. THE INTESTINES ( <i>Continued</i> ) . . . . .	173
Diarrhea ( <i>Continued</i> ) . . . . .	173
Acute Inflammatory Diarrhea (Acute Ileocolitis, Acute Dysen- tery) . . . . .	173
Descriptive Summary . . . . .	173
Etiology . . . . .	173
Lesions . . . . .	174
Course of Lesions . . . . .	175
Symptoms . . . . .	176
Urine . . . . .	177
Blood . . . . .	177
Summary of Course . . . . .	177
Diagnosis . . . . .	178
Treatment . . . . .	180
Medicinal . . . . .	181
Prophylaxis . . . . .	182
XVI. THE INTESTINES ( <i>Continued</i> ) . . . . .	184
Typhoid Fever in Childhood . . . . .	184
Descriptive Summary . . . . .	184
Age Incidence . . . . .	184
Etiology . . . . .	185
Pathology . . . . .	185
Symptoms . . . . .	186
Course . . . . .	190
Complications . . . . .	190
Diagnosis . . . . .	191
Differentiation . . . . .	191
Pneumonia . . . . .	191
Miliary Tuberculosis . . . . .	191
Treatment . . . . .	192
Prognosis . . . . .	193
Paratyphoid Fever . . . . .	195
General Consideration . . . . .	195
Etiology . . . . .	195
Bacteriology . . . . .	195
Lesions . . . . .	196
Symptoms . . . . .	196
Complications . . . . .	197
Diagnosis . . . . .	198
Prognosis and Treatment . . . . .	198

# CONTENTS

xv

## CHAPTER

PAGE

Chronic Ulcerative Colitis . . . . .	198
Descriptive Summary . . . . .	198
Etiology . . . . .	198
Lesions . . . . .	199
Symptoms . . . . .	199
Treatment . . . . .	201

## XVII. CHRONIC INTESTINAL INDIGESTION AND CELIAC DISEASE . . . . . 204

Chronic Intestinal Indigestion . . . . .	204
Treatment . . . . .	205
Celiac Disease (Infantilism with Fatty Stools) . . . . .	206
Nomenclature and History . . . . .	206
Etiology . . . . .	207
Physiology and Pathology . . . . .	208
Symptoms . . . . .	208
Course . . . . .	212
Complications . . . . .	213
Diagnosis . . . . .	213
Treatment . . . . .	214
Prognosis . . . . .	217

## XVIII. DEFICIENT APPETITE AND CONSTIPATION . . . . . 219

Persistent Anorexia Due to Asthenia . . . . .	220
Effect of Disease . . . . .	220
Perverted Appetite (Pica) . . . . .	220
Constipation . . . . .	221
In the Newly Born . . . . .	221
In the Breast-fed Infant . . . . .	221
Treatment . . . . .	222
In the Artificially Fed Infant . . . . .	223
Treatment . . . . .	224
Chronic Constipation in Children Past Infancy . . . . .	226
Definition . . . . .	226
Occurrence . . . . .	226
Mechanism . . . . .	226
Etiology . . . . .	226
Symptoms . . . . .	229
Diagnosis . . . . .	229
Treatment . . . . .	230

## XIX. INTESTINAL OBSTRUCTION AND MALFORMATION . . . . . 233

Etiology . . . . .	233
The Defect . . . . .	233
Symptoms . . . . .	234
Diagnosis . . . . .	236
Prognosis and Treatment . . . . .	237
Volvulus . . . . .	237
Intestinal Stone . . . . .	239
Fecal Impaction . . . . .	239

	Congenital Dilatation and Hypertrophy of Colon (Hirschsprung's Disease) . . . . .	239
	Congenital Microcolon (Small Colon) . . . . .	245
	Disturbances of Meckel's Diverticulum . . . . .	245
XX.	ACUTE SURGICAL DISEASES OF THE INTESTINES . . . . .	249
	Appendicitis in Childhood . . . . .	249
	Descriptive Summary . . . . .	249
	Nomenclature and History . . . . .	249
	Etiology . . . . .	249
	Anatomy and Pathology . . . . .	250
	Symptoms . . . . .	251
	Differential Diagnosis . . . . .	253
	Other Infections . . . . .	254
	Complications . . . . .	254
	Treatment . . . . .	255
	Prognosis . . . . .	255
	Intussusception (Invagination, Acute Type) . . . . .	255
	Descriptive Summary . . . . .	255
	Etiology . . . . .	256
	Anatomy and Pathology . . . . .	256
	Symptoms . . . . .	257
	Course . . . . .	258
	Diagnosis . . . . .	260
	Treatment . . . . .	260
	Peritonitis . . . . .	261
	Fetal . . . . .	261
	In Early Infancy . . . . .	261
	In Later Infancy and Childhood . . . . .	262
	Latent Peritonitis . . . . .	262
	Streptococcic Variety . . . . .	263
	Pneumococcic Variety . . . . .	264
XXI.	ABDOMINAL EXAMINATION AND CERTAIN DISTURBANCES OF THE	
	ABDOMEN . . . . .	267
	Abdominal Examination . . . . .	267
	Causes of Obscure Abdominal Pain . . . . .	269
	Visceral Ptosis . . . . .	270
	Abdominal Purpura . . . . .	273
XXII.	ABDOMINAL TUBERCULOSIS . . . . .	276
	Occurrence in Childhood . . . . .	277
	Etiology and Pathogenesis . . . . .	277
	Symptoms . . . . .	280
	Diagnosis . . . . .	282
	Complications . . . . .	283
	Prognosis . . . . .	284
	Treatment . . . . .	284
	General Treatment . . . . .	285
	Treatment of Special Symptoms . . . . .	285
	Surgical Treatment . . . . .	286

# CONTENTS

xvii

CHAPTER

PAGE

XXIII. ABNORMALITIES AND DISEASES OF THE RECTUM AND ANUS . . .	287
Congenital Abnormalities of the Rectum and Anus . . .	287
Prolapse of the Rectum and Anus . . . . .	289
Proctitis . . . . .	291
Gonorrheal Proctitis . . . . .	292
Fissure of the Anus and Rectum . . . . .	293
Hemorrhoids . . . . .	294
Polypus . . . . .	294
XXIV. INTESTINAL PARASITES . . . . .	295
Varieties . . . . .	296
Ascaris lumbricoides (Round-Worm) . . . . .	299
Necator americana (Hookworm) . . . . .	303
Trichocephalus dispar (Whip-Worm, Thread-Worm, Tri- churis) . . . . .	304
Tænia (Tapeworm) . . . . .	304
Amebic Dysentery (Infestation with Entamœba histolytica) . . . . .	305
Infestation with Lamblia (Giardia) intestinalis . . . . .	309
Infestation with Balantidium coli . . . . .	309
XXV. POISONING . . . . .	310
Accidental Poisoning . . . . .	310
Alkalies . . . . .	310
Acids . . . . .	311
Carbolic Acid . . . . .	311
Alcohol . . . . .	311
Arsenic . . . . .	311
Aspidium and Oil of Chenopodium . . . . .	312
Atropin . . . . .	312
Camphor . . . . .	312
Formaldehyd . . . . .	313
Lead . . . . .	313
Mercury . . . . .	313
Opium . . . . .	313
Santonin . . . . .	314
Phosphorus . . . . .	314
Silver Nitrate . . . . .	314
Turpentine . . . . .	314
Food Poisoning . . . . .	315
Botulism . . . . .	315
Alimentary Allergy . . . . .	316
Milk-Sickness . . . . .	320
XXVI. DISEASES OF THE LIVER, BILLIARY TRACT AND PANCREAS . . .	324
Physiology . . . . .	324
Disturbances in Early Life . . . . .	325
Tumors . . . . .	325
Catarrhal Jaundice . . . . .	326
Hepatic Insufficiency . . . . .	327
Benign Jaundice from Round-Worms . . . . .	328

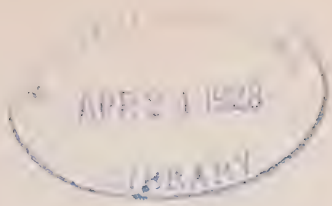


	PAGE
Gall-Stones (Cholelithiasis) . . . . .	328
Cirrhosis . . . . .	330
Fatty Disease of the Liver . . . . .	330
Hydatid Disease . . . . .	330
Diseases of the Pancreas . . . . .	331
APPENDIX . . . . .	335
Technical Methods in Pediatric Practice . . . . .	335
Roengen Ray in Gastro-intestinal Diagnosis . . . . .	335
Thick Feeding . . . . .	337
Sour Milk Feeding as a Therapeutic Measure . . . . .	338
Tube Feeding (Gavage) . . . . .	338
Methods of Water Administration . . . . .	339
Stomach Washing (Lavage) . . . . .	339
Proctoclysis . . . . .	340
Hypodermoclysis . . . . .	340
Intraperitoneal Injection . . . . .	341
Drug Dosage for Children . . . . .	342
Widal Reaction . . . . .	342
De Silvestri's Color Reaction in Typhoid Urine . . . . .	343
Test for Occult Blood in the Urine and Stool . . . . .	343
INDEX . . . . .	345

## ILLUSTRATIONS

FIGURE	PAGE
1. Enlarged protruding tongue . . . . .	5
2. Double harelip and cleft-palate with central snout-like protrusion containing one tooth . . . . .	7
3. Ectopia of the heart with harelip and cleft-palate . . . . .	8
4. Dental dam with tapes for use as an obturator . . . . .	9
5. The dental dam in place, child sucking the bottle . . . . .	9
6. Lesions on lips and tongue in herpetic stomatitis . . . . .	<i>Frontispiece</i>
7. Characteristic pursing of the lips due to increased salivary secretion . . . . .	28
8. Destruction of portion of cheek near the angle of the mouth due to gangrenous stomatitis . . . . .	34
9. Characteristic retraction of head and glandular swelling of neck on the affected side . . . . .	44
10. Drawing illustrating the site of the abscess . . . . .	45
11. Large multicolor salivary cyst of parotid in the newly born infant . . . . .	55
12. The deciduous teeth at eight months . . . . .	60
13. The deciduous teeth at fourteen months . . . . .	60
14. The deciduous teeth at eighteen months . . . . .	62
15. The deciduous teeth at thirty months . . . . .	62
16. Radiogram of upper jaw in young child . . . . .	63
17. The teeth at six years . . . . .	66
18. The teeth at eight years . . . . .	66
19. The teeth at ten years . . . . .	67
20. The teeth at twelve years . . . . .	67
21. Pulp development at six years . . . . .	68
22. Pulp development at twelve years . . . . .	68
23. Congenital absence of middle portion of the esophagus . . . . .	72
24. Upper portion of the esophagus . . . . .	73
25. Four open safety pins in the esophagus . . . . .	76
26. Stricture of esophagus following lye burn . . . . .	82
27. Insalivation of food through gastrotomy wound . . . . .	84
28. Radiogram in pyloric stenosis showing the barium still in the stomach at the end of four hours . . . . .	117
29. Contractions of the stomach showing the peristaltic waves in pyloric stenosis . . . . .	118
30. Nine-year-old girl with pyloric hypertrophy . . . . .	125
31. Appearance of infant during act of rumination . . . . .	131
32. Canvas cap used for rumination . . . . .	133
33. Quilted cotton jacket for the premature infant . . . . .	157
34. Section of the ileum in a case of membranous ileocolitis lasting two months . . . . .	175
35. Temperature curve in ileocolitis . . . . .	179
36. Extreme emaciation in protracted case of typhoid fever . . . . .	190
37. Celiac disease . . . . .	210

FIGURE	PAGE
38. Greatly enlarged colon in celiac disease . . . . .	211
39. Celiac disease . . . . .	212
40. Severe case of celiac disease showing extreme wasting . . . . .	213
41. Homemade corset belt for relief of constipation due to lack of abdominal tone and to visceral ptosis . . . . .	230
42. Front and back view of corset in place . . . . .	231
43. Older child with typical Hirschsprung's abdomen . . . . .	241
44. Large abdomen due to Hirschsprung's disease . . . . .	242
45. Perforation of Meckel's diverticulum . . . . .	247
46. Five-year-old girl with visceroptotic abdomen and slumping posture	271
47. Three-year-old Negro boy with ascitic type of tuberculous peritonitis	281
48. Typical temperature curve with morning remissions and evening exacerbations in tuberculous peritonitis . . . . .	282
49. In the center of this drawing a perforation of the rectal wall is seen .	289
50. Prolapse of the rectum . . . . .	290
51. Intestinal obstruction due to masses of <i>Ascaris lumbricoides</i> . . .	302
52. Photograph of thirteen-year-old child with carcinoma of liver . .	326



# DISEASES OF THE DIGESTIVE SYSTEM IN INFANCY AND CHILDHOOD

## CHAPTER I

### THE MOUTH

**Characteristics at Birth.**—In very young infants the examination of the structures of the mouth is difficult. There are several reasons for this: both the external opening and the internal circumference are small; the cavity of the mouth is practically obliterated by the thick, broad tongue which comes in contact with the hard palate and gums; much resistance is encountered when the spatula is introduced. It is necessary to use a broad, firm tongue depressor and a strong light in order to view the pharyngeal cavity.

The lining of the lips differs somewhat from that of later infancy, being thicker on the inner edge, the appearance of which has been likened to a third lip. The mucous membrane of the mouth is especially dry and hyperemic during the first few days of life while the child is losing weight and the intake of fluids is scanty. Even up to the time of dentition the mouth usually contains much less saliva than later. The salivary glands are present at birth, but they have less secretory function until the end of the second year, by which time they have increased many times in size.

During the newly born period most infants normally have a few small, hard nodules in the mucous membrane of the gums, roof of the mouth and median raphe of the palate. These nodules are yellowish-white in color, about the size of a mustard seed, and are anatomically small retention cysts of the mucous glands. They are known as Bohn's *epithelial pearls*. Absorption takes place in a week or two. No significance can be attached to their presence.

The surface of the tongue at the edges shows prominent papillæ and the center is covered with a grayish coat which is shed after a few days.

In addition to the peculiar structure of the mucosa of the lips and cheeks at this time of life, each cheek contains a deposit of fat enclosed in a connective-tissue capsule which assists in the mechanism of suckling. This *sucking pad* prevents the cheeks from being drawn between the gums. It is a wise provision of nature that this fat deposit is persistent in childhood in spite of the loss of adipose tissue elsewhere in the body during severe nutritional disturbances or other illness. The sucking pad stands out most clearly when the face is wasted from infantile atrophy (marasmus).



The infant of one month has as high a palate as the child of six to twelve months of age, but a little narrower. In an effort to find the normal shape of the palate at various ages, plaster models were made by Denzer, and measurements of the heights and widths were recorded. He used these to establish normal figures for comparison with the palates in diseased conditions of the tonsils and adenoids, and in rickets. It would be correct to assume that the deforming effects of adenoids and tonsils do not begin to affect the upper jaw during the first year of life. It is possible to watch for the beginning of characteristic changes in the palate as the criterion for the age at which adenoids, if causative, should be removed.

**Mechanism of Sucking.**—The organs of the mouth are adapted for nursing. The structures involved in this mechanism are the lips, gums, cheeks, tongue, hard palate, the soft palate and its projection, the uvula. Negative pressure is brought about during suction by withdrawal of the air content of the mouth into the lungs, and by the sudden enlargement of the cavity of the mouth by depression of the lower jaw and the contraction of the tongue. The cheeks are drawn in and the back of the tongue assumes a grooved shape and forms a trough for conveying fluids to the back of the mouth. There is also a trough formed on each side of the mouth between the cheek and the margin of the gums.

The nipple is held and squeezed by the anterior structures of the mouth. The pressure within the breast is maintained chiefly by the nipple, and is obviously somewhat less than the suction pressure of the average infant. The lips prevent ingress of air from the front, while air is excluded from behind by the closing of the nasopharynx at the isthmus of the fauces, which is assisted by the action of the soft palate, pillars and base of the tongue. The epiglottis guards the opening into the larynx. Fluid passes to either side into the pharynx and esophagus (Scammon).

The suction force of the baby can be measured by the manometer, and is found to be greater in the early months. Much more energy is required to empty the breast than the bottle; the latter, however, being dependent upon the size of the hole in the nipple. Vigorous suction stimulates the flow of milk from the breast, and increases the salivary secretion and the development of the muscles and jaws.

**Microorganisms.**—The mouth is sterile at birth, but soon develops bacteria from breathing and from nursing. The usual microorganisms in the mouth are harmless until some trauma or illness reduces the integrity of the mucous membrane. For this reason the mouth of the young infant is to be let alone and the practice of washing or swabbing never permitted.

## MALFORMATIONS OF THE STRUCTURE OF THE MOUTH

**Tongue-Tie.**—The most common, simple deformity in the mouth concerns the anterior insertion of the frenum. Some difference of opinion

exists as to whether so-called tongue-tie is to be considered a pathological condition and whether it is of any importance. The writer believes that it is of little significance.

The upper attachment of the frenum is normally well back on the under surface of the tongue. In tongue-tie a short frenum and folds of mucous membrane on each side unite the tip of the tongue to the floor of the mouth and the posterior, lingual surface of the gums. At birth the tongue itself is normally short, so that it is not always easy to determine how much the frenum interferes with its movements, unless it is attached to the point of the tip when it produces marked traction downward.

In the newly born the projecting part of the tongue is as yet incompletely developed, and the frenum may normally be well forward. As the infant grows older the front of the tongue goes on growing and becomes normal. Butlin and Spencer state that persistent cases of tongue-tie have never been seen by most surgeons. These writers regard the attitude toward this condition as a superstition of long standing.

As to a possible disturbance of function, I have never seen such an infant who failed to empty the normal breast, the nipples of which were well developed. It is emphatically stated by von Reuss that tongue-tie cannot interfere with nursing. When there is harelip and cleft-palate a freely movable tongue is needed in the attempt to produce a vacuum. If the tongue is still bound at its tip by the time the child is talking, it is possible that an indistinctness of speech may be occasioned thereby.

*Treatment.*—*Contra-Indication.*—Tongue-tie should be left alone, as there is no benefit to the child to be derived by clipping. If one severe hemorrhage or other accident occurs in the practice of the physician he will not thereafter regard the operation as trivial. Fatal hemorrhage has been not infrequently reported. The writer knows of one case, fortunately not in his personal practice, that died in spite of heroic efforts at hemostasis. The usual cause is the injury to the ranine arteries and veins. One must also remember that the newly born infant may bleed easily because of the disturbance in the bleeding and coagulation time. The literature contains many references to the meddlesome clipping and stripping of the tongue by midwives.

The parents are apt to place undue emphasis upon the importance of tongue-tie, and to insist upon clipping. The physician is apt to yield to the request. The family should be informed of the uselessness and the possible danger in the procedure. Infected ulcers and resulting scarring have been reported at the site of the wound, which resulted in a serious immobilizing of the tongue, a condition of much greater significance than the original one. Other ill effects have been noted, such as relaxation of the tongue with resulting tongue-swallowing, asphyxia, and hypertrophy of the organ (macroglossia). In looking through the literature one finds

not infrequent references to accidents, some of which were as long as two hundred years ago.

*Operation.*—If, in the opinion of the experienced observer, the frenum does interfere with function, its division should be done with strict precautions. It should always be preceded by an estimation of the bleeding and coagulation time. A hemorrhagic tendency is indicated when as many as five minutes are required for the needle stab in the heel or finger to stop bleeding or for the drop of blood placed upon the slide to show definite retraction.

In the seventeenth century Petit devised the groove director with the split broad handle for the purpose of straddling the frenum and preventing injury to the vessels on the tongue. This instrument is still in wide use. The tongue is raised and held, the frenum entering the notch in the end of the director. The cut is made below the instrument below the middle of the frenum which is simply nicked. The tongue should not be stripped. The movements of the tongue will usually complete the severance of the frenum by tearing or stretching its fibers. No after-treatment is necessary except the exclusion of dirty objects from the mouth.

If hemorrhage occurs, it is well to place a gauze pad under the tongue and to draw the tongue forward and secure it by a ligature placed through the tip and secured to the chin. The resulting pressure may be sufficient to stop the bleeding. Continued hemorrhage requires an anesthetic and the ligation of bleeding vessels. Injections of human blood may be of benefit in stopping the bleeding and in replacing what has been lost.

**Tongue-Swallowing.**—Abnormal mobility of the tongue may be due to malformation of the frenum or to other causes. The tongue may fall backward toward the pharynx and result in the so-called tongue-swallowing. This may persist as a habit and be productive of vomiting. In marked obstruction from the presence of the tongue in the back of the mouth cyanotic attacks may result. Courtin reported a case of this kind in which relief from the cyanosis was obtained by holding the tongue in its normal position with a spatula. This simple but tiresome measure was used for a long period until symptoms gradually disappeared.

The frenum may be too long, so that the tongue is not fixed in the mouth. The mechanism of tongue-swallowing is the backward turning of the tip until it reaches the esophagus, whereby the opening into the larynx is entirely covered. The abnormal mobility may result from complete division of the frenum.

**Tumors and Deformities of the Tongue.**—*Angioma* occasionally affects the tongue. It is characterized by the protrusion from the mouth of the greatly enlarged, reddish-blue mass made up of arteries, veins and capillaries. The appearance of the hypertrophied tongue at once suggests a birth-mark. The growth is painless and benign, but is unsightly. The size may vary from time to time with the amount of blood contained. The



function is disturbed. The mouth is dry and dirty, the swallowing and breathing impaired.

Such a growth is not amenable to surgical treatment because of the danger from hemorrhage. The use of radium is advisable. In case of obstruction to deglutition and respiration an operation becomes necessary.

*Papilloma of the Tongue.*—Warty growths are sometimes found upon the dorsal surface of the tongue, due to hypertrophy of the papillæ. They may be congenital or develop in childhood and later life. The growth may be single or multiple and may form a thick mass. Papillomata need not be removed in childhood but may be left until adult life.

*Congenital Hypertrophy.*—Macroglossia, as a symptom of idiocy, is found especially in cretinism and mongolism.

No relief in the size or protrusion of the tongue can be expected, from any treatment employed, in the mongolian idiot. Some benefit has been obtained in the cretin by the early use of thyroid extract. This should be begun at birth. The difficult diagnosis of hypothyroidism in the early months of life makes it improbable that much will be done in reducing the size of the tongue in such infants.



FIG. I.—ENLARGED PROTRUDING TONGUE  
A common manifestation in mongolism. The above children are twins, one of whom is a typical mongol and has the characteristic tongue.

The typical form of macroglossia is due to lymphangioma, owing its enlargement to a dilatation of the lymphatic spaces. It may be large from birth or begin as a small birth-mark of the tongue and later begin to grow. Certain cases seem to have followed clipping of a tongue-tie.

Hypertrophy of the tongue from lymphatic malformation shows the disturbances of function which result from the size and obstruction. Surgical measures or treatment with radium should be employed.

*Microglossia* (small development), *aglossia* (absence), *bifid* and *double tongue* are rare congenital deformities occasionally found mentioned in the literature.

Congenital fissures or furrows are sometimes present on the surface of the tongue. They are not inflammatory or ulcerative, and have an epithelial lining. The cause and significance of the malformation are not known, but it is possible that they may have been due to fetal inflammation from syphilis. In the absence of syphilis they are of no importance although a somewhat similar appearance is found on the surface of the tongue of the mongolian idiot.

**Cyst.**—Congenital cysts have been rarely encountered. One case of a cyst at the base of the tongue soon caused suffocation and death.

**Harelip and Cleft-Palate.**—Harelip is an easily recognized defect, and when unaccompanied by cleft-palate must be regarded as deforming but not a particularly difficult malformation to repair. A wide gap in the hard palate associated with defects in the lip and nostril presents a most serious problem in the welfare of the individual. No congenital deformity of importance is so frequent as harelip; while defects in the palate make up the second most common type.

Because of the relative frequency, the impairment of nutritional function and the disfiguration from the combined deformity, the question of proper and timely treatment demands careful and experienced consideration. If uncorrected, the health and future happiness will be markedly disturbed. In any event the malformation is a pitiable one. Plastic surgery has done much for this affliction by restoring function and leaving little deformity. The physician who first sees the child is confronted with the problem of maintaining nutrition and of deciding upon the time for surgical intervention.

*Occurrence.*—These defects are found once in about one thousand births. The incidence of harelip or cleft-palate in native Swedish children was 130 in 12,500. Occasionally there is more than one child affected in the family. Of the above-mentioned 130 cases, 2.3 per cent showed an hereditary tendency, and 3.7 per cent had a similar deformity in other children in the same family (Edberg). The possibility of children being born with a deformity is a matter of serious concern, especially when the family already possesses an affected child. When both sides of the family have a history of harelip or cleft-palate, the appearance of a like deformity in the offspring is much more likely than when it can be traced to only one side. In all published figures boys are the more frequently affected.

There is simply a failure of growth and union between the lateral and central portions of the lip. The cause is unknown, but occurs in early fetal life. In palatal defects the two bony processes (palatal plates) which form the lateral halves fail to close at some part or all of their extent, and therefore do not fuse into a median raphé.

*Disfiguration.*—The simplest defects in the union of the structures of the mouth are seen in cases of bifid uvula and in the central dimpling of the chin. These cause no cosmetic or functional disturbance. The next degree is that of a small grooving of the upper lip, through the skin and mucous membrane, with a scar running upward to one nostril. Stone has called attention to the asymmetry of the nostril in even the mild cases of harelip. Other cases have more extensive defects, with notching on one or both sides of the median line beneath the nares, or rarely with an absence of the central portion of the lip, mild notching of the alveolar process of the upper jaw, and deformities of the nose. The disfiguration is frequently increased by a central island of lip with a forward position of



the intermaxillary bone, so that it resembles a snoutlike mass. All degrees of deformity of the face and cheeks may be present in extreme cases, but the lower lip and jaw are seldom affected.

Cleft-palate of various degrees may exist both with and without hare-lip, but the most serious complications occur with their coincidence. The palate varies in the degree of involvement from a cleft uvula to a separation of the palatal plates of any width. The sides may be either symmetrical or irregular. The defect may extend forward through the entire roof of the mouth. The oral and nasal cavities are continuous. The septum of the nose and the intermaxillary bone may be unattached or joined to the palate on one side.

*Effect upon Health.*—The inability of the infant with severe degrees of cleft-palate and harelip to take adequate amounts of food soon results in definite nutritional losses. The physician at once faces the problem of maintaining health, vigor, and even life. The cases with the greatest defects are the most difficult to feed. Early functional restoration becomes a matter of necessity in such cases. However, attempts at repair are often disap-



FIG. 2.—DOUBLE HARELIP AND CLEFT-PALATE WITH CENTRAL SNOUT-LIKE PROTRUSION CONTAINING ONE TOOTH

pointing and unsuccessful, and the infant goes through a protracted period of repeated operations. Foote has recently called attention to the marked underweight of infants coming to operation, due to the difficulty in taking food and to the wastage of much that is offered the child. The infant consumes a long time in nursing and becomes fatigued, especially on the breast, where more energy is required to extract the milk than from the average bottle. It is impossible for the infant with cleft-palate to produce the negative pressure in the mouth necessary to empty the breast, as air enters the mouth directly from the nose or the lip defect. The breast soon dries up.

Infection in the nose or mouth may result in respiratory complication. Stone speaks of the risk of pneumonia because of the abnormally large opening in the nose and mouth which prevents the warming and moistening of the inspired air. A poor nutritional state and marked weakness lower

the resistance to general infection. Intestinal infection may occur from the derangement in feeding, from the weakened state of the child or from secondary infection in the nose. Food enters the nose and may act as an irritant. It is difficult to keep infection out of the nasal sinuses.

Operation introduces other risks to the child. A certain amount of shock may result from the operative procedure. Infants in poor condition do not stand operation well. Infection of the field is a frequent result, whereby the child is still further endangered and the stitches may slough out. Pyelitis is a frequent sequel of cleft-palate operations.

*When to Operate.*—Much difference of opinion exists as to the best time for operation. The decision must be made by the consideration of the



FIG. 3.—ECTOPIA OF THE HEART WITH HARELIP AND CLEFT-PALATE

This infant had not only a cleft in the lip and palate, but also one of the sternum through which the heart protruded. Other bodily malformations are sometimes associated with congenital deformities of the mouth.

immediate and ultimate welfare of the individual case. Some operators have made it a rule to begin the restoration of the defects within the first week of life before infection and nutritional disturbances have begun. The following factors should be considered:

1. *The ability of the infant to secure by natural or artificial means sufficient food for the body needs.* Malformation to the extent that suckling is prevented makes it necessary to feed with a spoon, dropper or tube. Tube feeding is best, as it insures a definite amount reaching the stomach, and there is less danger of contamination and infection of the nasal cavity. Irrespective of the apparent success of food introduction, nutrition and health may become impaired.

2. Delay in operation allows a *still further widening of the defect* by the contraction of the facial muscles. As the infant grows older the parts become firmer and more ossified, and the approximation of the palatal edges more difficult by operation.

3. Older infants usually bear operation better, but the *state of the nutrition* has more to do with successful operation and good results than any age factor.

4. The most favorable time for operation depends somewhat upon the degree of the palatal, nasal and lip deformity. Mild cases in good condi-

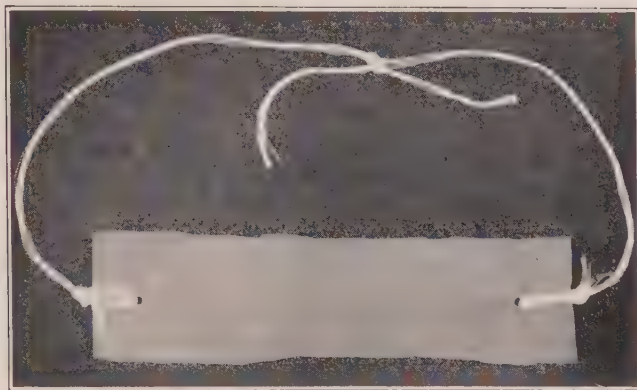


FIG. 4.—DENTAL DAM WITH TAPES FOR USE AS AN OBTURATOR  
(Courtesy of John A. Foote and *Am. J. Dis. Child.*)

tion can be safely operated upon at once, or postponed until later infancy. Marked defects interfere with food intake and with the integrity of the



FIG. 5.—THE DENTAL DAM IN PLACE, CHILD SUCKING THE BOTTLE  
(Courtesy of John A. Foote and *Am. J. Dis. Child.*)

tissues, so that nothing is gained by waiting if there is no hope of improving the general condition of the child.

5. *Respiratory infections* interfere with convalescence. Anemia of the infant impairs the operative results and prognosis.



6. An experienced operator in this field will take all factors into consideration when deciding upon the time for repair in the individual case.

*Treatment.*—The preoperative treatment is important in improving the health of the child and in obtaining the best local conditions in the operative field. If the infant is strong, healthy, not infected, and has a good state of nutrition, repair may be made at once.

As a means of improving the general state of the child, the effort should be made to produce a gain in weight. Insufficient intake of breast or bottle milk is best supplemented by the use of concentrated foods. Milk is boiled down to half volume and the nutritional value increased by the addition of well-cooked thick cereal such as farina or cream of wheat. In case the breast cannot be nursed, the milk is expressed and added to the cereal mixture, or fed with a tube. Tube feeding is advisable when by other methods much food is wasted or the infant fails to gain.

When the lip is intact, or has been repaired, the application of a strip of dental rubber dam over the nose while nursing blocks the entrance of air and enables the infant to take a larger amount of fluid. The use of such an external obturator has been suggested by Foote, who found that the increased food intake results in a better maintenance of nutrition in the infant. The obturator consists of a piece of rubber dam, two by four inches, placed over the nose and held in place by tapes which go around the head. The rubber is lifted from time to time so that the infant may breathe better.

The general health of the child should be maintained by fresh air and sunshine, cod-liver oil and iron.

The nasal cavity and mouth should be kept clean and free from infection. Irrigation with water and the application of a mild antiseptic solution should be used after each feeding.

Freedom from nasal or general infection, such as colds, is a necessity for obtaining the best results from operation. There should be no inflammation in the region to be repaired.

*Prognosis.*—In addition to the anatomical results, the functional and cosmetic outcome is of great importance. Not the least of the functional factors is the prevention of speech defect. The ideal to be obtained is that the speech should be normal and without nasal quality. The earlier that operation is done, with due regard to the safety of the child, the less will be the possibility of abnormal speech. Monnier has advised the early beginning of speech training.

The cosmetic results mean much to the happiness of the individual in later life. The symmetry of the nose and lip is of chief importance. It is to be expected that the labial scar will be more or less noticeable. Finally, the gap in the teeth makes it necessary that dental closure should be attempted, both for cosmetic and functional reasons.

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## CHAPTER II

### DISEASES OF THE MOUTH

#### THE LIP

**Herpes of the Lip.**—Herpetic lesions of the mucocutaneous border of the lips are of frequent occurrence in childhood. The condition is popularly known as fever blister or 'cold sore'. A group of somewhat painful small vesicles suddenly appears. These are ruptured by scratching, followed by exudation of serum. A crust forms which may bleed easily by irritation.

The exciting cause is unknown, but has some relation to fatigue, loss of sleep and to febrile conditions. It is a frequent accompaniment but not a symptom of upper respiratory infections. It occurs in some cases of pneumonia and meningitis.

The lesions heal rapidly if left alone, but children can with difficulty be kept from removing the crust with their finger nails. For this reason restraint of the hands is necessary. Application of collodion or dry boracic acid may hasten the healing process.

#### THE FRENUM

**Simple Ulcer.**—Simple ulcer of the frenum may be present during whooping-cough. At one time it was considered frequent enough to be regarded as a symptom of pertussis, but it is of only accidental occurrence, due to the friction produced in the paroxysm of coughing. The lesion is superficial and is overlooked in many cases. It may also be found in any poorly nourished infant who has chronic cough or a gastro-intestinal disease. Ulcer of the frenum is seldom seen in the toothless infant, therefore it must owe its origin to mechanical causes, such as the irritation of the lower surface of the tongue and frenum by the lower incisor teeth. It is said to be especially liable to develop when these teeth have been erupted prematurely. Trauma is necessary for its inception and in this respect and from the appearance of the lesion it is similar to the herpetic lesion of stomatitis.

The treatment consists in the use of mild washes of borax or salicylic acid solution, and in persistent cases the application of tincture of iodine, or the nitrate of silver once daily. As an accompaniment of whooping-cough it is of no consequence and disappears spontaneously.

**Riga's Disease.**—Riga's disease is a type of ulceration seen in infants exclusively. It does occur rarely in this country but is said to be common

in Italy. I can recall one case in my experience. The location and characteristics of the disease are so peculiar that once seen it is never forgotten. A papillomatous or fibromatous growth the size of a pea develops in the frenum of the infant. The beginning is probably inflammatory. There is at first a small white thickening of the frenum, later becoming red and larger. The summit of the growth undergoes superficial ulceration and necrosis and becomes covered by a pseudomembranous exudate, which has been designated as diphtheroid subglossitis. It affects infants from six to eighteen months of age. Slight interference with nursing is the only disturbance from the lesion. The growth disappears under the local treatment of the ulceration. This consists of silver nitrate solution applied daily, or of curettement in obstinate cases.

## THE GUMS

**Gingivitis.**—Swelling of the gums just previous to the eruption of teeth may cause slight fever, irritability, restlessness at night, failure to eat because of impaired appetite or tenderness, and the inclination to keep the fingers in the mouth. There is definite edema or inflammation of the gums. When the gum is markedly swollen and the outline of the tooth plainly visible, but not through, some cases will be benefited by rubbing the gum with a clean spatula or spoon or by making a crucial incision. Often the wound from lancing a gum heals with no immediate result in the appearance of the edge of the tooth. In such a case the resulting scar may add to the difficulty of the later eruption. The indication for lancing becomes less urgent the longer one practices among children, but there is benefit in relieving the tension in the edematous, transparent gum which is slow to yield to absorption.

**Abscess and Periostitis.**—The regular inspection of the teeth will reveal one of the most common diseases in childhood—the presence of abscess and fistula. The early superficial lesion is the so-called gumboil. The gum is swollen, bulging, red, painful and shows evidence of a collection of pus. The adjacent surface of the cheek may be tender and swollen. The surface of the gum becomes perforated and a discharging sinus persists. Rarely the abscess breaks into the antrum or through the cheek. The underlying lesion is the alveolar abscess, accompanied by necrosis of the jaw in neglected cases. It is most commonly seen as a result of infection of the deciduous teeth, in which caries has been active.

Fever may be present when the inflammation is acute, when absorption of pus is abundant. Such cases may show other general symptoms of toxemia, and be mistaken for tuberculosis or typhoid fever.

Children go for months with draining abscesses, unknown to themselves or their parents, or treated with indifference. Especially among the poorer classes one sees instances of swollen jaws which have been bandaged for

days or weeks, with no attempt at treatment other than external application. By the time an abscess has formed, little can be done except drainage through rupture or incision. Extraction will eventually be necessary.

**Hypertrophy.**—Secondary hypertrophy may occur from irritation from carious teeth. The resulting increase in size is known as polypus of the gums.

Primary hypertrophy may begin in childhood or in early adult life. There is some doubt about whether it is ever congenital. Usually both jaws are affected, on one or both sides. The growth overlies the alveolar border of the jaws. The hypertrophied tissue is firm, fibrous and of normal color. There may be other physical defects and in children mental impairment is usually noted. The growth is considered to be fibromatous. The teeth are seen to be nearly buried in the gums. There have been several cases in children, observed especially by British physicians. Three cases among children of one family are mentioned by Heath.

The jaws may be so large as to interfere with closing the mouth, and older children are sensitive about their appearance. The muscles of the face give the appearance of being tense, the lips are turned outward, and the mouth is large. No pain or symptom other than the deformity is present.

The treatment is surgical, requiring radical excision of the diseased tissue. It necessitates the removal of the teeth and alveolar periosteum in reaching all the hypertrophied tissue. Hemorrhage is the chief operative complication.

**Scurvy.**—The characteristic oral changes in scurvy are found in the gums, but the salivary glands and the teeth are also affected, but in a less conspicuous way.

A hemorrhagic inflammation of the gums is the pathological change. They bleed readily, especially when touched, are swollen, spongy and may cover the teeth. As a rule there is a dark bluish discoloration of the gums, which is followed by ulceration. Hemorrhages are not common until after eruption of the teeth. Scurvy may be latent for several months before mucous membrane lesions appear.

The teeth themselves lose their luster, turn yellowish, get loose in their sockets and may be broken off. The salivary glands are said to be atrophic during the disease, and the secretion of saliva decreased. The appetite is impaired. Vomiting may occur and intestinal indigestion is usually present. The disease is most apt to occur during the age when the incisor teeth are appearing, and the gums of these, especially the upper, are most frequently affected. Before the tooth is erupted, the gum resembles a large bluish vesicle filled with blood. The gums are painful and irritated by the presence of food. The mouth closely resembles ulcerative or mercurial stomatitis.

The history is of definite value in the diagnosis. Usually some proprie-

tary food such as a powdered malt food will have been in use for months with no fresh article in the diet. It is well in all cases of hemorrhagic and ulcerated gums to feed orange or tomato juice, not only for their nutritional but for their diagnostic value.

## THE TONGUE

The older physician placed great emphasis upon the appearance of the tongue as a diagnostic aid. In the days before bacteriology and pathology became correlated with the clinical study of disease, reliance had to be placed chiefly upon the physical appearance of accessible regions of the body.

In childhood especially the abnormal conditions of the tongue are chiefly those of local diseases of the mouth. Scarlet fever, inflammatory and ulcerative states show definite diagnostic appearances in the tongue. Dryness, abnormal coating and sordes are indicative of febrile and infectious states, but are not sufficiently limited to any one disease, such as typhoid, whereby a specific diagnosis may be made.

**Geographic Tongue.**—Superficial desquamation of patches of epithelium, with an ever-spreading margin is frequently seen on the dorsum and edges of the tongue. Under the names of *lingua geographica*, *annulus migrans* and *glossitis areata exfoliativa* this easily recognized but poorly understood phenomenon has been voluminously discussed. It has been incorrectly ascribed to psoriasis or regarded as the lingual manifestation of eczema.

The occurrence is usually in children between six months and four years of age, though it may be present at birth or throughout childhood. The cause is not known. Parasitic attack would naturally be first suspected, but investigations have not given any evidence which can be considered conclusive. Czerny has advanced the opinion that epithelial desquamation is connected with the eczematous or exudative diathesis. It is frequently seen in children thus affected, but as often in those who have not had eczema.

*Appearance and Character of the Lesions.*—The characteristic appearance of the common type is due to the coalescence of crescent-shaped figures upon the surface of the tongue, which results in patches resembling somewhat a geographic map. These may change their location from day to day. Some of the surfaces are grayish-white, and others somewhat reddened. The grayish parts represent the early stage of the lesion, caused by hyperplasia of the epithelium, and the later redness is due to the subsequent loss of the superficial layer of epithelium, leaving the papillæ prominent. Around the edge of the desquamated area is a somewhat thickened margin, representing the remains of the grayish-white hyperplasia. The lesion begins as a small whitish round spot on the border or tip of the tongue, and the well-defined margin moves forward in ring-like curves toward the center and back of the tongue. In the spreading of the patch one area may fuse with



another. Repair of one part of the tongue takes place while another is becoming affected. The condition is not related to stomatitis. Other types of desquamation are described, of shorter duration, and of like insignificance.

*Symptoms.*—There is no functional disorder connected with this manifestation. Pain is never present, the secretions of the mouth are normal. The affection is chronic and may last for years.

The condition is sometimes mistaken for the mucous patches of syphilis. To one who is familiar with both lesions there is little difficulty in the differentiation.

*Treatment.*—None is effective or necessary. Mild washes such as boric acid are often used, but without results other than to effect the cleanliness of the mouth. When the child has any disturbance of the health, attention should be paid to the general condition. Those children who have an exudative diathesis should receive suitable dietary and hygienic care.

**Glossitis.**—Inflammation from infected wounds does not often occur. The most severe degree of glossitis results from the accidental ingestion of strong acid and alkali solutions, which may produce necrosis of more or less depth. The tongue is more or less inflamed in severe scarlet fever and measles, during the course of intestinal diseases, and also accompanying ulcerations of the mouth. In the neighborhood of ragged or decayed teeth the edge of the tongue often shows marks of thickening and inflammation, which may result in ulcer formation.

With the exception of deep burns or wound infection, the treatment of glossitis is unnecessary. The comfort of the child is increased by cold drinks.

Edema and swelling of the tongue from the ingestion of food to which the child is sensitized occurs suddenly as a manifestation of allergy. Such a phenomenon is not an inflammatory process, and subsides with the use of an emetic, laxative, or a hypodermic injection of adrenalin.

## THE JAW

**Necrosis.**—Necrosis of the upper jaw has been reported in newly born and older infants. Paunz has reported seven cases. The disease is an osteomyelitis originating from infection of the maxillary antrum through the nose.

The symptoms are edema and abscess of one side of face, pus from the nostril on the affected side, swelling of the periosteum of the alveolar process of the upper jaw, and resulting fistula. The disease may remain local, but occasionally general sepsis may develop and death result.

The outcome is good in localized cases; drainage and removal of necrotic bone tissue being indicated.

**Tumor.**—Congenital tumors in this region are always benign. They are of rare occurrence in the newly born.

Epulis is a slowly growing tumor of the jaw which sometimes occurs in childhood. According to Scudder, the growth is on the border line between an inflammatory process and a neoplasm. It originates from the periosteum of the jaw or beneath the mucous membrane. The cause is irritation from an incompletely extracted tooth root. When it becomes malignant the growth resembles a sarcoma.

In the beginning the process may be mistaken for a gumboil. It is at this period that diagnosis is the most valuable, when removal can be more complete and less difficult.

**Sarcoma.**—The effect of trauma in producing neoplasm of the jaw is now recognized as positive in many of the cases. Sarcoma of either jaw occurs in childhood, but less frequently than in an equal period in adult life, as shown by the figures from various writers and from the Massachusetts General Hospital (Scudder). About 8 per cent of the cases are found between one and ten years of age. The early removal of the growth by curettage may obviate the necessity of resection of the jaw.

## SYPHILIS OF THE MOUTH

Acquired syphilis is seldom seen in children. It is found in infancy due to infection from an infected wet-nurse, or in older children by transmission from drinking-cups or from kissing. Fortunately such accidents occur rarely, but prophylactic care should be constantly exercised. Chancre of the mouth differs in no way from the primary lesion seen in adults. The later manifestations are likewise comparable.

Secondary syphilitic lesions of the mouth in children are nearly always due to untreated hereditary infection. Seldom is any other organ of the mouth than the lips invaded, the typical lesion of which is the linear or radiating fissure (rhagades) or the characteristic scar which follows its recovery. There is a splitting of either lip, at the junction of the skin and mucous membrane. The fissures are usually multiple and radiating. In their active period the discomfort and pain are considerable. After healing there is marked disfiguration due to the retraction and loss of substance beneath the mucosa. In my experience the lips are seldom attacked at the corners of the mouth, but usually in the middle third. Chronic linear ulceration confined to the angles of the mouth is not syphilitic, but is due to a pyogenic infection known by the French as *perlèche*.

Condyloma may occur on the mucous membrane just inside the corner of the mouth. Still reports such a lesion appearing as early as two weeks after birth. He has also seen a lesion on the buccal mucosa resembling catarrhal stomatitis. The mucous membrane is grayish-white, and appears as if there had been a recent application of silver nitrate. The lesion is superficial and never thickened as is the mucous patch of syphilis.

Inflammation of the tongue (glossitis) is found in about 4 per cent of



congenital syphilis (Still). The lesions are either thickened or ulcerative. They vary in appearance, some have a leathery thickness, others are dry and brown, and some are moist (mucous patch). The ulcers are oblong and have a clear-cut edge.

Syphilitic lesions of the tongue are seen during the first two years of life. Fissure of the tongue may be a symptom of hereditary or acquired syphilis.

A defect in the border of the soft palate near the base of the uvula is sometimes seen in children. The depression or notch is due to the loss of substance in the epithelium or mucous membrane, and is 1 or 2 millimeters in extent. It occurs on both sides of the uvula. It is of congenital origin and, according to Tanturri who has described it, is due to syphilis. Other evidence of specific infection should, however, be found before making such a definite diagnosis or beginning treatment.

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## CHAPTER III

### DISEASES OF THE MOUTH (*Continued*)

#### STOMATITIS

**General Consideration.**—Diseases of the mucous membrane of the mouth are classified under the name of stomatitis. The term is applied to affections which have such a localized lesion as a simple ulcer, as well as to all grades of parasitic and microbic invasions and inflammations of the lips, gums, cheeks, palate and tongue. Some of the qualifying terms aptly denote the type of disease, while others are inappropriate and have been the cause of much confusion. This confusion has arisen from the attempt to designate the disease by a single type of lesion, by the lack of knowledge as to the etiology, and by loosely using the same name for different diseases.

The secretions and the mucous lining of the mouth harbor an abundant variety of microorganisms. These may come from without, or multiply favorably within the oral cavity. These microorganisms are usually harmless, but pyogenic varieties are also present. Stomatitis at its onset may be due to organisms normally resident in the mouth, but more specific types may contribute to the later course of the infection. With an uninjured mucosa, infection does not occur. One exception might be made to this rule, *i.e.*, in the case of an epidemic of thrush which sometimes spreads to all the newly born infants in a hospital nursery. The method of dissemination in such cases is obviously through contamination of rubber nipples, or of spoons, fingers and other articles which reach the well baby's mouth. I have twice seen epidemics where no trauma or mouth washing had been practiced. However, it is to be remembered that the resistance of the mucosa in the newly born is low, due to the dryness and hyperemia of the mouth.

The only common varieties of stomatitis in children with a proven specific microbic cause are thrush and the ulcerative-gangrenous type. In severe cases staphylococci, streptococci and probably other organisms may play a complicating rôle.

#### THRUSH

Thrush is a well-known parasitic disease of young infants, involving the mucous lining of the mouth with the formation of characteristic small white flakes or patches. It is the type that is peculiar to the newly born, and occurs epidemically in hospitals where young children are closely

grouped. It appears to a mild extent in many healthy babies. It occurs commonly as an extensive involvement of the mouth, and in young infants, especially the bottle-fed, who are suffering from febrile, digestive or nutritional disturbances.

**Etiology.**—The cause of thrush is a fungus belonging to the genus *Oidium*. The organism consists of two forms, one of which is yeast-like (spore or unicellular) and the other is thread-like or filamental (mycelium). The form depends upon the character of the medium in which the parasite grows. The organism was discovered by Berg in 1839, but it was named *Oidium albicans* by Robin a few years later.

Infancy is a predisposing factor in that the incidence is most common during the second week of life, especially when the child is housed in an institutional nursery where the disease may already exist.

The infective parasite is introduced into the mouth in various ways. It is now agreed that the mother may have the fungus upon her nipples, in her mouth, and even in the vagina (Noack). From the latter source the child's mouth may be contaminated at birth. The most probable mode of transmission is by the mother's nipples, but it is evidently carried also by dirty fingers, rubber nipples, and other objects put into the infant's mouth. The fungus may remain latent in the secretions or confined to small areas somewhere in the oral cavity, to become active when general or local resistance is lowered (trauma).

An injured mucosa is necessary for the initiation of the disease. However, the mouth of the newly born is predisposed to thrush infection, because of the delicate structure of the mucosa and its dryness and hyperemia during this period. Occasions for traumatizing the mouth were formerly quite common, but are now prevalent only among the uninstructed families.

**Lesions.**—*Distribution.*—Thrush attacks the pavement epithelium of the mucous membrane. The most common sites are the inner surfaces of the cheeks, the tip and edges of the tongue, the lips and the hard palate. It may also affect the gums, the fauces, pharynx, and rarely the esophagus, stomach and intestines. There have been reported cases of extension to the parotid and submaxillary glands. When the disease attacks the larynx and lungs it is probable that the infection travels through the blood stream. General infection is said to have occurred.

Unusual distribution of lesions and occurrence in older children have been reported recently. Schlutz saw two chronic cases occurring in the same family. They presented features which are unusual in thrush. The disease lasted for years and always exhibited a tendency to diarrhea. The children had a temporary loss of the hair and nails.

Christison described a case in which the disease had persisted for over two years when it appeared upon one finger and on the back of the neck. The scrapings from the skin showed that the reddish, branny scales contained the *Oidium albicans*. Much of the skin all over the body became

involved, and the child died of pulmonary thrush infection, three and one-half years after the appearance of the disease in the mouth.

A rare manifestation of infection by the fungus of thrush was found on the back of the tongue, where a chronic indurated ulcer had been present in an undernourished infant (Wilkins and Bayne-Jones). The ulcer was small, as hard as cartilage, the surface dirty-gray, and the edges undermined. The examination of the material scraped from the ulcer showed the characteristic yeastlike bodies and threads. Successful treatment resulted from the application of nitrate of silver.

**Symptoms.**—There may be no premonitory or early symptoms suggestive of an infection of the mouth. Sufficient inflammation of the mucous membrane will, however, interfere with the appetite. The infant may attempt to nurse and because of sensitiveness to the presence of food refuse to continue. If nursing causes discomfort, it may be found that feeding with a spoon is acceptable. Marked pain is absent unless the disease has spread to the throat, where it is excited by swallowing. Vomiting sometimes occurs and renders the feeding more difficult. Thrush is a serious complication when occurring in the poorly nourished infant, as it affects the appetite and digestion.

Looseness of the bowels is present, and may be troublesome. The stools are green, highly acid and irritating, causing excoriation of the skin of the buttocks.

These disturbances make the child restless and unhappy. There is usually no elevation of temperature.

The inspection of the mouth shows at first general redness of the mucous lining. In a few hours numerous small flecks of adherent white deposits are noticeable. These resemble tiny milk curds, but it will be found that they cannot be easily wiped off, and if removed a raw bleeding surface is left. The lesions in the beginning are round. They are at first found on the tip and edges of the tongue, inside the lips, on the mucous surface of the cheeks not far from the corners of the mouth, and a few upon the gums. A spreading of the size of the lesions may cause a coalescence until a layer of white deposit is more or less continuous throughout the mouth. The soft palate and the posterior vault of the pharynx are usually spared.

After a few days the lesions become yellowish-brown, dry and fall off. In private practice among robust infants the disease tends to rapid recovery in a few days. When neglected, or roughly handled, especially in the poorly nourished child the disease may become chronic and intractable.

**Diagnosis.**—The diagnosis is easy. It is often first discovered by the mother, who may be depended upon to explore the regions of the gums and tongue of her infant. The occurrence is rare in the vigorous breast infant, but is to be suspected in the young artificially fed baby who is not thriving on its food.

Other varieties of stomatitis, with the exception of erythematous inflam-



mation, are rare at this time of life. True herpetic lesions do not occur. Diphtheria is frequently mentioned in the differential diagnosis but it is not to be considered in the absence of the disease in the nose or throat.

**Treatment.**—*Prophylaxis.*—In hospital practice especially where there are numerous infants it is important to prevent the spread of the disease to others. The bottles and nipples should be boiled after each feeding, and kept from contamination thereafter.

The nipples of the mother should be washed before and after nursings, using soap, water and a mild antiseptic solution.

*Curative.*—Gentleness and the avoidance of unnecessary manipulation in the mouth will be found to be as important as the local treatment.

The sucking bag suggested by Escherich has been efficient in my experience. The infant, for one-half hour between feedings, is allowed to suck a pad of fresh gauze or large folded handkerchief which is wet in an alkaline solution. Borax, boric acid or bicarbonate of soda, one drachm to the pint of water, thus used, comes in contact with the affected mucous membrane without producing the trauma that may occur with other methods of local treatment. The oral cavity is kept alkalized for more or less continuous periods in this manner.

Alkaline solutions may also be introduced by the pipet. One part of borax in ten of glycerin is recommended by the French writers.

Local application of gentian violet solution has been recently suggested. It is used once daily in 1 per cent solution.

The digestion, nutrition and general health of the infant must be given attention.

**Prognosis.**—Simple thrush recovers promptly with persistent local treatment and hygiene. The general health of the infant is not affected by thrush unless there is underlying nutritional or digestive disturbance. The spread of thrush to regions other than the oral cavity is a serious matter.

#### CATARRHAL STOMATITIS

Simple inflammation of the mucous lining of the mouth is characterized by local or general redness and swelling, some pain, an increase in the secretions of the mouth, and later by the development of a milky or light yellow color of the epithelium, especially of the gums and tongue. No other type of lesion is present in simple catarrhal or erythematous stomatitis, but it should be remembered that a similar inflammation of the mucosa may precede or accompany other diseased conditions in the mouth.

The etiology suggests a mild infection, and such is probable, though in the presence of so many microorganisms in the mouth, it is impossible to isolate the one which may be responsible. As suggested by Cooley, this type of stomatitis may be due to a variety of organisms. While erythema is present in the newly born, the mouth is dry, and true catarrhal stomatitis is

seldom found. The disease, which is characterized by an increase in saliva, seldom appears before the eruption of teeth. Various factors lead to the inflammatory development. During dentition the affected gums show a varying degree of such changes. If the process becomes extensive, other factors exist, such as poor nutrition and lowered resistance. Traumatism or other mechanical irritant may be causative. A definite but fortunately not frequently observed type of inflammation is due to the accidental ingestion of irritant poisons. Scarlet fever, when severe, and measles are generally accompanied by oral congestion. Any febrile or digestive disturbance may lead to irritation of the mouth.

**Symptoms.**—Catarrhal stomatitis may be the unsuspected cause of a mild indisposition in the older infant or child, who has begun to be restless and irritable. A routine inspection of the mouth should be made a part of all examinations in childhood, a necessary procedure which is sometimes overlooked. The appetite is poor, the child does not enjoy food because of discomfort from the effort of eating, and because the taste is impaired. The stools are apt to be loose in the infant, and constipated in the older child.

The nature of the disturbance may be suspected when the lips are seen to be swollen and when saliva is pouring from the partly opened mouth. There is no offensive odor to the breath in this type of stomatitis. When the oral cavity is inspected there is found unusual redness and some swelling of the gums, especially at the gingival margin. The region of the newly appearing teeth alone may be involved. The teeth are almost hidden by the surrounding gums, which may be covered by a cloudy deposit which can be easily rubbed off.

The mucous membrane is congested and bleeds easily. In a good light the mucous follicles show elevation, and their secretion is increased. The inner surface of the lips is also involved in the redness and swelling. The tongue is red at the edges, and the papillæ are abnormally prominent. There is marked coating of the dorsum of the tongue, and the accumulation of debris during long-standing febrile diseases produces sordes and a cracking of the tongue surface and the lips.

In typhoid fever, severe scarlet fever, and other serious infections, the stomatitis, once developed, may not subside until the end of the febrile course. In simple stomatitis the manifestations are of short duration and the mouth is usually well within five days.

**Treatment.**—Spontaneous recovery is the rule in the mild cases, and it is not probable that what is usually done in the way of sporadic local treatment hastens the recovery. The comfort of the child may be increased by the frequent giving of teaspoonfuls of shaved ice. Applications which cause resistance and struggling on the part of the child may produce trauma and increase the restlessness sufficiently to contra-indicate their use.

Because of the fact that catarrhal inflammation may be the forerunner



of ulcerative and more serious lesions, and in the markedly sick child the mouth does not so readily recover its normal condition, the mouth and teeth should be kept clean, *fresh* peroxid of hydrogen being efficient. It is well to have the child hold in the mouth as long as possible a mild antiseptic solution of boric acid, or the alkaline antiseptic solution made with the Seiler's tablet (1 in 2 ounces of water). If there is a tendency to marked swelling and bleeding, alum water (1 drachm to the pint) may be used to rinse out the mouth.

#### HERPETIC STOMATITIS

##### *(Maculofibrinous or Follicular Stomatitis)*

Herpetic stomatitis occurs commonly from six months to five years of age. There may be no apparent cause or it may appear in the course of exanthematous or other infectious disease. Lesions are found typically on the tongue and lips, where they will be seen as well-defined, more or less round, ulcerations covered with a yellowish-white deposit and surrounded by a reddish zone.

**Etiology.**—Numerous attempts have been made to establish a bacterial causation, but as yet nothing has been accepted as probable. There is such a wide variety in the number of lesions, and so few symptoms are caused thereby, that any constant bacterial infection seems improbable. A single ulcer is occasionally found on the mucosa of the cheek, lip, or faucial pillar which is apparently accidental and without any significance. This should be regarded as simple, idiopathic ulceration and not as a part of the whole clinical entity under discussion. In some cases the mucous lesions accompany herpes on the lips and skin, and if seen early the grouping is characteristically striking.

The cause of herpes is not known. Indigestion, febrile and toxic states and some disturbance of the local nerve filaments in the skin are explanations which have been long used. Catarrhal inflammation often precedes and a circumscribed amount usually accompanies the disease. Uncleanliness probably plays as much part here as in other types. The dirty habit of sucking fingers, pacifiers and soiled candy is usual among children. The common occurrence during the time that the deciduous teeth are appearing would indicate that the child gets more dirt into the mouth at such a time, has less resistance during that period, or that the teething process itself has something to do with the incidence of the disease. It would seem that this form of stomatitis, especially when accompanied by fever, is an infectious process, but probably a secondary one.

I have found a concept prevalent among physicians that oral ulceration is only a part of a similar process extending throughout the intestinal tract. This opinion seems to have arisen as an explanation of the digestive disturbances which some cases show, particularly the tendency to diarrhea. Only in dysenteric disease is ulceration of the intestine common, and there is no

constant or primary stomatitis. When the two coexist the ulcerations in the mouth result from a cause other than that of the intestinal infection.

**Symptoms.**—Usually there are not over fifteen or twenty ulcers throughout the mouth, while a much smaller number, from three to five, will sometimes be found. The lesions may be isolated or grouped. The lips, the cheeks, the corners of the mouth, the lower part of the gums, the tip and edges of the tongue, are the favorite sites. There is more or less difference in opinion as to the early appearance of the lesions, probably due to the fact that these have disappeared by the time the case is seen, but some erythematous or catarrhal inflammation is present. It is a matter of no importance whether there is always a preliminary grayish-white vesicle formation or whether there is simply a mild necrosis of the superficial epithelial cells, since the resultant lesion is always a shallow ulcer which heals without any scarring or deformity. The outline of the ulcer is round and marked by rather definite edges. The surface is covered with a yellowish-white, curd-like coating. Outside of the border is a definite area of redness and inflammation, which is in marked contrast with the dull coating within the lesion. The lesions vary in size from  $\frac{1}{16}$  to  $\frac{1}{4}$  of an inch. The smallest size is found when the lesions occur in clusters, following which several may coalesce to form a larger patch. It is because of the complication with herpes on the skin that the lesions of mucous membrane have received the name of herpetic. There may be dozens of the glistening watery blisters on the cutaneous surface of the upper and lower lips, while on the tongue at the tip or on the inner surface of the cheeks will be seen a similar group of rather small lesions, probably  $\frac{1}{8}$  of an inch in diameter. A good light is necessary to view the exfoliating epithelial layer and the reddened areola around the lesion (see Frontispiece, *i.e.*, Fig. 6).

Children may be little disturbed by this affection. There is no fetid odor to the breath. As a rule the child is somewhat sluggish and the appetite is poor. The temperature may be slightly above normal. As is true with other types of stomatitis the disposition is markedly irritable. This is not surprising as the sleep is restless, the child fatigued, and the nutrition temporarily arrested. At times there is diarrhea.

Most of the discomfort, however, comes from the local conditions in the mouth. The raw surfaces are further irritated by the presence of food, which causes burning sensations and pain. Usually the lymph glands are somewhat inflamed.

The disease lasts from one to two weeks during which time the child suffers somewhat in weight and well-being because of the failure to eat sufficiently. The duration is considerably longer in the poorly nourished child and adds to the weakness and body loss, especially if there are relapses.

Smears should be made from the lesions when they occur near the margin of the gums, and if Vincent's organisms are found the specific treatment for that type of infection should be employed.

**Treatment.**—It is interesting to note the treatment in England twenty-five years ago. Bu'llin and Spencer, in a monograph at that time, recommended the giving of a laxative and the rinsing of the mouth with water, following which they state that the child gets well. However, when the patient was quite sick, a laxative was given every hour. They advised 4 or 5 grains of potassium chlorate internally every four hours, that the tongue be wiped with borax water, and in case of sluggish ulcers that silver nitrate or alum be used as an astringent. With the exception of the unnecessary amount of laxatives, the foregoing treatment has been little improved upon by anything which has been suggested since, and is much the same as employed at the present time.

Rinsing the mouth with *fresh* peroxid of hydrogen in an equal amount of water should be done several times daily, followed by an irrigation of the mouth with a solution of potassium permanganate (1 : 1500). These agents will help to keep the mouth clean and tend to prevent relapses and complications, even if they do not hasten the disappearance of the ulcers.

It is a common practice to touch the ulcers of the lips and tongue with a 5 per cent solution of silver nitrate once daily. This need be done, however, only when the lesions are persistent. Holt recommends that a bleeding surface be rinsed with a solution of burnt alum. In most cases the family usually finds it difficult to care for the child, so the **treatment in mild cases** should be simple and painless. Severe cases will be best handled in the hospital where the physician and nurse can be more successful in the local treatment and in the feeding of the child.

The primary cause has recently been assigned by Gerstenberger to metabolic or nutritional disturbance, with a secondary invasion by parasites resulting in the ulcer formation. He found that orange juice and yeast when added to the diet rapidly cured herpetic and ulcerative stomatitis, and herpes labialis. He believes that a dietary deficiency is a factor in favoring the causation of oral inflammation. It is possible that the administration of foods containing water-soluble vitamin B brings the nutrition of the oral tissues back to their normal state so that the micro-organisms find an unfavorable medium. The use of such a simple and necessary addition to the diet is advisable in all cases, and should be tried.

The feeding of these children is difficult. Nourishing food, preferably liquid, such as milk and sweetened fruit juices, vegetable soup, and thin cereals should be given in as large amounts as the child can be induced to take. When diarrhea is present the use of protein milk rather than sweet milk is preferable. The recovery from a predisposing disease hastens the return of the oral condition to the normal.

#### ULCERATIVE STOMATITIS

The most important type of stomatitis as regards the seriousness of the lesions, the danger of transmission to others, and the importance of a



correct diagnosis with specific treatment is that caused by infection with the organisms of Vincent. The features by which the disease is chiefly recognized are the necrotic and bleeding tendencies of the gums, the putrid odor of the breath, the marked accumulation or dribbling of saliva, and the presence of the Vincent organisms in the material swabbed from the diseased area. This type of infection never appears before the presence of teeth.

**Nomenclature.**—Although due to the same organism, ulcerative stomatitis should not be confused with Vincent's angina (sore-throat) which involves the tonsil in a pseudomembranous and ulcerative inflammation. The popular term "trench mouth" was much in use among the troops during the recent war for this condition.

**Etiology.**—The disease is an infection with the spirillum and fusiform bacillus which are forms of the same organism. These may have been latent or present in the mouth or may have recently been transmitted from external sources. Other members of the family may harbor the infective agents in diseased gums, and communicate them by kissing or by the contamination of utensils used in cooking and eating.

According to Haden the usual habitat of the organisms is between the teeth in the pockets which form under the edge of the gums. Food and other débris are seldom removed from these regions, and the anaërobic organisms thrive beneath the gums. When the teeth are loose the food fills the spaces and the underlying region is suitable for harboring the organism. Uncleanliness and neglect of the teeth and gums are predisposing causes, but the disease will seldom if ever develop without an underlying condition of lowered vitality. This may be from weakness, fatigue, infectious or nutritional diseases. What may be the predisposing cause in the numerous cases which develop in an epidemic is difficult to state.

**Lesions.**—The lesions in childhood are acute. Infection begins under the edge of the gums or along the roots of the teeth. The gums are purplish-red, definitely swollen from edema or inflammation. The edges are friable, spongy, red and bleed easily. As soon as the necrotic process develops the gum line becomes ragged and gangrenous. By direct contact the disease may spread to the lips and cheeks opposite the diseased gums, which are likewise inflamed and may contain superficial grayish ulcers of considerable size. The teeth become loosened, cases are reported in which the roots are exposed and the teeth come out, but this is now undoubtedly rare. I have never seen a case of such severity, probably because of the early recognition and efficient treatment. The same necrotic process may extend to the alveolar portions of the jaw bones.

The primary ulceration is a necrotic process in the gums, at first of a superficial nature and then extending to the deeper layers. A grayish coating of putrid necrotic mucous membrane overlies the ulceration, and consists of broken-down tissue fibers, epithelial and blood cells.

The disease usually begins in the gums of the lower front teeth, and may spread to part or all of the gingival region of both jaws, at first along the dental margin.

**Symptoms.**—The writer has been impressed by the diagnostic appearance of the facial expression in these children. The picture is one of marked misery, and there is a characteristic pursing of the lips, due to the accumulation of saliva in the anterior part of the mouth. When the lips are separated there is drooling of saliva in a steady stream.



FIG. 7.—CHARACTERISTIC PURSING OF THE LIPS  
DUE TO INCREASED SALIVARY SECRETION

There is much pain and discomfort due to the swelling and acute disease of the gums. The attempt to have the child open the mouth for inspection, treatment or feeding is resisted because of fear, tenderness or pain being constant. Peevishness and crying are frequent.

One of the most noticeable characteristics is the foul breath which comes from the putrefactive necrotic regions.

When the mouth is opened the swollen lips bleed easily, and saliva pours out. The child seems to have difficulty in separating the upper and lower jaws. The appearance of the mucous surfaces of the front of the mouth is striking, and in severe cases gives the impression of irreparable damage. Portions of the gums are swollen so as to nearly cover the teeth, while at places on the margin the gums are ragged, necrotic, and covered with a dirty grayish deposit. The surfaces tend to bleed easily, always when touched and often spontaneously. Purulent bloody débris oozes or may be expressed from the edges. Ulcers of various sizes and necrotic inflammation may be found on the lips, cheeks, underneath the tongue, or upon the structures of the fauces. The tongue is swollen and heavily coated.

The course is marked by sudden onset, malaise, loss of appetite, and by fever of some extent, usually below 102° F. The submental and submaxillary lymphatic glands are palpable and tender. The health of the child suffers from the disturbance, particularly from the restlessness and loss of nourishment. In children who have previously been ill this infection mark-



edly adds to their poor condition. Untreated cases may persist for many weeks. Under early proper treatment the disease seldom lasts over ten days. Sequelæ are absent, with the possible exception of deformity in the margin of the gum which may be shrunken. Neglected cases in which the disease goes on unchecked may result in necrosis of the dental sockets. Death from severe and extensive involvement has occurred, but is now rare.

**Diagnosis.**—The local appearance of ulcerative stomatitis may resemble that of pyorrhea alveolaris. Pyorrhea is a very chronic disease with, as a rule, few acute manifestations. The gums show the same tendency to bleed and there is usually a purulent discharge oozing from the dental margin. It is generally considered that pyorrhea is in no way connected with Vincent's organism.

Mild cases are apt to be confused with other types of stomatitis, especially catarrhal and herpetic. It should be remembered that ulcerative stomatitis is primarily a gingivitis, and that the necrosis of the gum margin is its early and chief characteristic. Morgan has well called attention to the complicating ulcers of a different type, which one may ascribe to a coincident infection from other causes, probably from secondary invasion with microorganisms whose virulence is increased by the poor resistance of the mouth. Incomplete results from the specific treatment of ulcerative stomatitis should suggest the presence of a complicating or different type of lesion in the mucous membrane.

**Treatment.**—*Prophylaxis.*—Of first importance is the maintenance of clean mouths in all the members of the household. The teeth and gums of all the children and of the parents should be kept free of dirt, food remains, and disease. Regular visits must be made to the dentist. The general health and nutrition of the children are to be guarded at all times. When acute illness occurs, better care than usual should be taken of the teeth and mouth. The tooth brush, and other means (dental floss, etc.) for cleaning between the teeth and around the gums should be used after each meal. Rinsing of the mouth with peroxid of hydrogen, followed by a few mouthfuls of chlorate of potassium solution is a good preventive measure.

When some member of the family has an unhealthy mouth, the spoons, glasses, etc., should be boiled before allowing others to use them. The child should be prevented from using the common drinking cup.

*Curative.*—Two measures are preëminent in the curing of ulcerative stomatitis. These are the local use and injection of preparations of arsenic. For application to the mouth the best preparation is a dilution of Fowler's solution as follows:

R	Liq. potass. arsenitis . . . . .	3.5 c.c.	ʒi
	Vini ipecac . . . . .	3.5 c.c.	ʒi
	Glycerin . . . . .	3.5 c.c.	ʒi
	Aq. mentha pip . . . . .	q.s. 30.0 c.c.	ʒi

This is gently applied with a fresh cotton swab to the diseased gums, tongue, lips and cheeks, every six hours. Paterson, at the Great Ormond Street Hospital, London, has been using this treatment in a large number of cases and recommends it highly. Because of the poisonous character of this mixture, he advises its dilution, using 15 drops in 2 drachms of water, so as to prevent the swallowing of too much arsenic. This diluted mixture may be put on the tooth brush or used for rinsing the mouth.

In severe cases and when treatment is begun late, the use of injections of neo-arsphenamin has been highly satisfactory. The result in cleaning up the Vincent organisms is spectacular. I have been using either the sulpharsphenamin preparation subcutaneously or the neutral neo-arsphenamin intramuscularly. From one to three injections are necessary. The dose is 0.1 gram in younger children, up to 0.3 gram for the older ages. The drug is dissolved in from 1 to 3 c.c. of sterile distilled water. Most cases need no other treatment than this.

As an adjuvant to the foregoing remedies the time-honored potassium chlorate is of value, when in addition to the Vincent's gingivitis there are other types of ulcers on the tongue or upon the inner surface of the cheeks. These are not due to the Vincent organisms and are not benefited by arsenic. The amount of potassium chlorate for a young child in twenty-four hours is 12 grains, and for children from two to five years 18 grains. This drug sometimes causes indigestion and should not be given at meals.

If the foregoing remedies are administered, cure will result in from three to five days as a rule. By such thorough medication relapses which are otherwise common will be prevented.

The child should be given plenty of nourishing food as soon as he is able to take it; the general condition should be improved by fresh air and sunshine as well.

#### GANGRENOUS STOMATITIS (*Cancrum Oris, Noma*)

This rare and fatal gangrene of children is now known to be due to infection with the Vincent organisms, which, beginning in the mucous membrane, extends to the entire wall of the cheek. The infrequency of its occurrence in recent years has made it difficult to adequately study the disease in the light of our present knowledge. Ulcerative stomatitis and noma are now considered by most writers as identical but differing in degree and severity. The infective agent is identical in the two diseases: in both there is first the invasion of the mucous membrane of the mouth, and a somewhat similar necrotic process. Certainly the clinical course of gangrenous stomatitis is entirely different from ulcerative stomatitis, probably due to the anatomical peculiarities of the location.

The general term of noma will be found throughout the literature applied to gangrenous infections not only of the mouth, but of those other orifices

having similar structure. The word has more historical interest than appropriateness, and dates back to the early Greek writers. Its original meaning was "feeding" or "spreading."

*Historical.*—Gangrene of the face has been well known since the earliest medical writings were recorded. Hippocrates and Galen mentioned noma, which, however, in the early times was made to include gangrene of any part of the body. In the seventeenth century the disease began to be accurately described by the Dutch and Swedish physicians who, however, were in ignorance of the cause. The disease was prevalent in Holland at that time and seemed to be usually a sequel of eruptive diseases and scurvy. Many writers noticed that the occurrence was almost entirely among poor children who were insufficiently fed and who dwelt in damp, unhealthy surroundings. The disease in America seems to have appeared first in an asylum for children in Philadelphia in 1819 and Coates writing in 1826 stated that the disease recurred each winter with disastrous results. He had collected seventy cases for his report.

**Etiology.**—The age at which this disease occurs is most commonly between two and five years, although some infants and older children have been victims. That it has been considered a disease of childhood is shown by the name sometimes used, "necrosis infantilis." The predilection for this age is chiefly due to the susceptibility to infectious diseases. When intestinal and contagious infections spread through the poorly equipped hospital of earlier days, epidemics began to be reported and the disease more frequent. Until recent years isolation and hygiene were unknown in the care of the hospital child. Advances in the methods of cleanliness, the knowledge of the care of the mouth during health and illness, reduction in the incidence of typhoid and other severe infections have all worked toward the lowering of the morbidity. It is probable that few of the younger physicians have ever seen a case. Most of the cases that I have known were in the early years of my practice and were among institutional children who were debilitated from dysentery or typhoid fever.

From the rarity of the disease one must conclude that the infection is seldom transmitted to another child and the existence of epidemics of the facial type of gangrene have occurred seldom if ever during the last hundred years.

The disease is never primary but is found in individuals who have been debilitated by a previous disease. Measles is the cause in fully one-half of the cases (Osler). Typhoid fever, whooping-cough, mercury poisoning, smallpox and scarlet fever were next in order of frequency as reported by Tourdes in 1848. At that time typhoid fever and smallpox were common manifestations.

Not only the lowered resistance and vitality of the child are necessary for the development of the infection, but the unhealthy and uncleanly state of the mouth in those seriously sick is a predisposing cause. Other



forms of stomatitis may precede it, and it is most probable that the disease has been an extension of an ulcerative stomatitis. Severe cases of the latter were formerly not separated from gangrenous conditions in other parts of the mouth. There is much similarity between ulcerative stomatitis and scurvy. The French, especially at one time, thought that noma and scurvy were closely associated.

The history of the bacteriology of this disease is a long one. Nearly every organism has been studied in reference to its possible relation to noma, a fact which is not surprising from the great numbers found in the mouth. In 1888, long thread-like organisms were found in the zone of advance near the necrotic tissue. Seiffert probably was the first bacteriologist to grow the spirillum in culture made from cases of noma. Since that time many observers have confirmed the etiological importance of the fusiform bacillus and the spirillum. Vincent, Plaut and Bernheim found the organisms in the ulceromembranous disease of the tonsils. The consensus of opinion is that the spirillum and fusiform bacillus are simply different stages in the life of the organism, and belong to the spirochætæ.

**Lesions.**—The beginning of this gangrenous disease is in the mucous membrane of one cheek, not far from the angle of the mouth. It first appears as a dark red swollen patch upon which soon develop small vesicles. The deeper tissues are infiltrated and infected. There is edema of the connective tissue, degeneration of the muscles and destruction of the myelin sheaves of the nerves. Changes occur in the walls of the blood-vessels. The lesion is essentially a tissue necrosis due to the overwhelming presence of microorganisms. It has been regarded by some as due to blocking of the circulation, but it is more probable that the destructive action of the Vincent organisms produces the changes. All the tissues of the cheek undergo necrosis and the gangrenous area steadily widens. The affected tissues are dark red and eventually black. Within the circle of destruction the region becomes a putrid mass with a gangrenous odor. Much of the surface of the face may become involved, sometimes surrounding the mouth and appearing in the opposite cheek. The underlying bones and teeth may be exposed and suffer necrosis.

The seven cases beginning in the auditory canal, reported by Holt, followed chronic otitis media which was being treated with a syringe found to be contaminated. The maxilla and the bones of the face in the region of the ear may be laid bare.

In addition to sepsis, the condition may terminate with abscess of the lung or pneumonia.

**Symptoms.**—A gangrenous odor to the breath easily calls attention to a marked disturbance in the mouth. This may occur during the course of infection or soon afterwards when it will be noticed that the child is not getting along well. There is definite pallor of the entire face until the disease reaches the skin, when it will be found that the color is violet or

greenish, the skin tight and somewhat oily in appearance. A mass is felt in the wall of the cheek about the size of an almond, but this is not painful.

When the mouth is inspected, there is seen a livid color of the mucous membrane, but as soon as ulceration occurs, the appearance is grayish, due to necrosis. The mouth may be filled with fetid bloody saliva. The disease proceeds rapidly through the tissues of the cheek until the skin is reached. The skin turns dark and finally black and is dry and parchment-like in consistency. The gangrenous area or eschar is seen upon the most prominent part of the swelling. The line of advance is marked by a red demarkation. The sloughened area may come away. The amount of tissue destruction is increased as the neighboring gums and other structures of the jaw are attacked. No other disease of the face is so ghastly. When a portion of the cheek wall has sloughed out, the teeth and jaw may be seen through the opening.

The general symptoms of the child vary markedly. Some cases are reported in which the children remain on their feet and play until the last day; other children are soon greatly disturbed by the illness. In the former type many cases have been reported without any fever except when a complication occurs. Temperatures of 103° F. or more are present in those children who are definitely sick from the start. Reports in the literature show that some children retain their appetite until the last, while others refuse their food. The thirst is marked. Vomiting does not often occur, but diarrhea is the rule.

Writers have called attention to the marked pallor of the face which persists until the last. They speak of the loss of expression which becomes noticeable. The eyes may show an edema, but as the end approaches they are markedly sunken. The tongue is moist, yellow and is said to be occasionally necrotic on the side near the involved cheek.

The child may lie quietly, or in other cases be peevish and fretful. When prostration occurs it is severe, the pulse becomes fast and difficult to feel. The disease is usually painless. As death approaches the restlessness is succeeded by apathy and exhaustion. The average duration of the disease is from eight to fourteen days. Practically all cases end with pneumonia. Gangrene may be carried to other parts of the body, and this forms the most serious complication. Gangrene has been reported in the lungs, pharynx, esophagus and stomach. Hemorrhage is rare.

**Diagnosis.**—The diagnosis is easy after the characteristic odor of the breath and a mass in the wall of the cheek have appeared. The other features which are important are the grayish appearance of the mucous membrane, the discoloration of the skin and the rapidity of tissue destruction. No disease other than carbuncle or anthrax is apt to be mistaken for noma. These, however, begin on the skin surface and produce deep ulceration.

**Prognosis.**—So many different conditions have been included under the name of noma that one doubts the reports of cure in even the small percent-



age of cases. The mortality is 100 per cent in series reported by some men. I have recently known of two children surviving the disease. The deformity of the face and the interference with speech and deglutition may be very great, and these tend to make the child's life intolerable. Some cases have been reported in which a certain amount of repair has taken place.



FIG. 8.—DESTRUCTION OF PORTION OF CHEEK NEAR THE ANGLE OF THE MOUTH DUE TO GANGRENOUS STOMATITIS  
(Courtesy of Harry C. Berger, Kansas City.)

**Treatment.**—The early recognition of the disease is most important in order that the use of arsphenamin may be at once begun, and if no benefits result therefrom, early excision of the diseased tissue be done. Extensive cauterization of the wound was formerly practiced. In spite of the earlier methods, the children died; so that one is warranted in trying the specific intravenous injection of neosalvarsan. So far as I know few cases have been reported in which this therapy was employed. The foul

condition of the mouth requires frequent rinsings, and this is best done with hydrogen peroxid.

**Prophylaxis.**—Prophylaxis is of far more use than treatment. It consists in regular care of the mouth during health and disease, especially when stomatitis has been present. In the presence of ulcerative or gangrenous stomatitis the utmost precaution should be taken to prevent the spread of infection to others. This is done by complete isolation and aseptic nursing. Dishes and linen are to be disinfected and boiled.

#### ULCERATION OF THE HARD PALATE

This is generally called Bednar's aphthæ, *ulcera pterygoides*, and by Epstein, "ulceration of the palatine angles." The site for the ulceration is on the median raphé or on one or both sides. The tendency to injury over these palatine eminences is probably greater than elsewhere in the roof of the mouth. The mucous membrane is especially vulnerable and tightly adherent to the hard palate. When injured it shows little tendency to heal. Young infants who are "afflicted" with mouth washing may get a mechanical abrasion of the epithelium. This disease affects mostly newly born infants, but it may occur in older children who have dry mouths from febrile or nutritional disturbances. The bad habit of sucking the tongue or thumb, or the pressure of nipples and pacifiers upon the roof of the mouth are chronic sources of irritation to the mucosa. Unclean conditions in the mouth also favor the production of epithelial damage. The rough use of the tongue depressor is liable to produce trauma in this region. From the fact that this disease and thrush are often coincident in the newly born infant's mouth, they may both be due to the same organism.

One or more ulcers will be found on the hard palate, usually on both sides of the median line, but in some cases connected by a lesion in the central raphé. The ulcer is superficial, about 1 centimeter in diameter, covered by an adherent yellowish-white deposit, attempt at the removal of which leaves a bleeding surface. The tissue change in this form of ulceration is an infectious necrosis of the epithelial layers. The deposit shows the usual desquamated cells and great varieties of bacteria. It is not probable that microorganisms have any more than a secondary place in the development of the lesions, although infection and sepsis may take place in emaciated or congenitally weak infants. Fränkel thought that the mucous membrane lining the roof of the mouth is in a vulnerable situation and is predisposed to lesions by the local lack of resistance to microorganisms. The ulcers are sensitive and on this account the infant may not take the nipple well. The course is usually short after proper treatment has been instituted.

The treatment is preventive. Friction, or pressure, from foreign objects in the mouth should not be allowed. Most infants suck the fingers

or the nipples on bottles for long periods of time. Fortunately the artificial pacifiers are not so much in evidence as formerly. An infant who has cleanliness of the mouth, whose mucous membrane is not injured by washing or by any other form of trauma, and whose nutrition proceeds normally, will not get this disease. A weak solution of silver nitrate is painted upon the ulcer once daily, and an alkaline solution of borax dropped into the mouth after each feeding.

#### MERCURIAL STOMATITIS

Stomatitis is most apt to occur when mercury is given by inunction, especially if it is applied in the region of the genitals or to an open skin lesion. Mercurial poisoning from accidental ingestion is of grave significance and involves all of the digestive tract.

Lesions in the mouth (rare in infants before the teething age) are due not only to previous infection of the gums, but to trauma of the gums, tongue and cheek by food and by the teeth. There is a systemic poisoning with changes in the blood and circulation. When the mercury is given in large doses with the object of causing salivation, there is great probability of local lesions occurring. Bessessen states that mercuric sulphid is precipitated in the cells of the capillaries, predisposing to a lowered vitality of the tissues.

*Symptoms.*—The disease is not seen before the eruption of teeth. The mucous membrane of the mouth is inflamed. The gums are swollen, red, soft, sensitive and retracted. Large, irregular, necrotic ulcers occur on the inside of the cheeks and on the edges of the tongue, produced by friction against the teeth. The teeth become loose, grayish-black and decay easily. The breath is offensive, the saliva increased and drooling. Swallowing is difficult because of the edema of the pharynx. Ulceration and sloughing of the gums occur from infection or trauma.

The treatment is the immediate withdrawal of mercurial therapy, the establishment of oral cleanliness, the use of potassium chlorate internally and locally.

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## CHAPTER IV

### AFFECTIONS OF THE PHARYNX AND TONSILS

#### THE PHARYNX

The pharynx is described as that part of the upper expanded region of the digestive canal between the soft palate and the esophagus. It has little to do with digestive processes, but is highly important in its conveyance of food. Its derangements directly concern the function of eating and nutrition. Malformations, paralysis of its structures, and serious inflammation of its walls affect the digestive system, primarily by interference with deglutition. Secondly constitutional disturbances exert a profound influence upon the digestion. Loss of appetite, insufficient intake, febrile and toxic states, vomiting, diminished gastric and intestinal secretion, constipation or diarrhea are common accompaniments of disease in the pharynx.

**Anatomical Characteristics.**—In infancy the shape of the pharynx is much different from that in the five-year-old child. The roof and the posterior wall form only a slight curve, in contrast with the marked angle which develops in later life. The pharynx grows in height and width but not in the anterior posterior diameter during the first five years (Scammon).

At birth the roof of the pharynx shows the beginning of the mass of lymphoid tissue which eventually becomes the pharyngeal tonsils, and which when hypertrophied is known as adenoid tissue. It is not until the sixth year of life that the adenoid growth has reached its maximum and begins to recede.

In the young infant the faucial tonsil is higher and nearer to the median line. The long diameter of the tonsil is horizontal, while in later childhood the tonsils are placed vertically. Their size increases rapidly in the first two years of life. Routine inspection of the tonsils during the first nine months shows little change from the appearance at birth, and seldom do they exhibit any hypertrophy. The tonsils are practically never inflamed in early infancy.

*Congenital obstruction* of the pharynx may cause respiratory embarrassment in the newly born. One such abnormality is a lingual thyroid at the base of the tongue (New). A flaccid tongue and palate may cause backward sinking of the tongue, so-called tongue-swallowing, and resultant difficulty in swallowing food (Butlin and Spencer).

**Foreign Bodies in the Pharynx.**—After infancy foreign bodies are occasionally found in the pharynx. Fishbones are the most common of



these. They are easily discovered but not always readily extracted. Small round objects such as beans or beads are often pushed into the nose and may remain for some time in the rhinopharynx.

In the routine examination of the throat, the writer once saw a medium-sized glass marble in the tonsillar fossa, snugly held by the anterior and posterior pillars. The mother recalled that the boy had "swallowed" a marble several months before, and nothing more had been thought about it.

**Disturbances of the Uvula.**—The function of the uvula in early life is largely that of assistance in closing off the fauces during the nursing act. It is generally considered to act as a "trapdoor" in preventing the entrance of food to the larynx, either in swallowing or during vomiting. That it does not always do this is obvious. Its involvement in the edema and swelling of quinsy is probably responsible for nasal regurgitation of fluids so frequently noted, especially on the affected side. Much edema remains after tonsillectomy for several days, especially following local anesthesia, and in severe tonsillitis the uvula may be swollen as a part of the catarrhal process. Membranous formation covers the uvula after the second or third day in untreated cases of diphtheria. Vincent's angina may likewise extend to the soft palate.

The uvula is sometimes inadvertently amputated during tonsillectomy. However, this accident is not productive of any harm to the child.

Bifid uvula is a congenital malformation, which must be considered in the nature of a mild degree of cleft-palate. It is productive of no known functional disturbance.

*Elongated Uvula.*—The size of the uvula varies considerably. The average length is usually under 2 centimeters. An elongated uvula may be 3 or 4 centimeters.

The abnormality begins probably in fetal life, so that it is relatively long at birth, and grows either at the usual rate or rapidly, due to the stimulation by attacks of acute inflammation. The pathological increase is only in the mucous membrane.

When the mouth is opened the elongated uvula is seen lying upon the base of the tongue, sometimes sticking to a pillar or doubled upon itself. The tip is much thinner than the body, is mucous and somewhat transparent.

The symptoms are slight or unimportant. Occasionally gagging or vomiting is caused in the susceptible child, and coughing may be excited in the croupous or asthmatic subject. Older children may complain of tickling or the sensation of a foreign body.

*Treatment.*—In the absence of symptoms the uvula should be left alone. After tonsillectomy the resulting contraction of the posterior pillars will usually raise or shorten the uvula, so that clipping is unnecessary. Marked hemorrhage is unknown in the absence of a bleeding tendency in the individual if the operation be skillfully performed.



**Acute Pharyngitis.**—Catarrhal inflammation of the pharynx is of frequent occurrence in children. When primary it is accompanied by more pronounced constitutional manifestations than in later life, probably due to the fact that no partial immunity has as yet developed. The febrile reaction is often out of proportion to the apparent mildness of the inflammatory changes. Fluctuation from 101 to 104° F. may be present for three or four days. The disease when secondary is most often epidemic and due to influenza, scarlet fever or measles. When there is a scarlet erythema of the pharynx accompanied by fever and vomiting, a scarlet rash can be expected within a day. Involvement of the pharynx always occurs in extensive diphtheria. A focus of infection in the nasal sinuses, adenoids or tonsils seems to predispose to pharyngitis, so that children may have frequent attacks, the cause of which is often ascribed to outdoor exposure. The frequent occurrence of pharyngitis in the winter months can only be explained on the basis of epidemicity, due to infection with some one of the microorganisms associated with ordinary colds. In some instances digestive disturbances, such as vomiting and constipation, seem to have a causal relationship. Febrile diseases which cause dryness of the mouth and pharynx favor the attack by microorganisms, as does also the overheating and dryness of the air in unventilated rooms during the winter.

The appearance of the mucous membrane is that of redness, dryness, moderate swelling, and the occasional presence of adherent mucus and pus. The lymph follicles in the posterior pharyngeal wall are sometimes noticeably enlarged and reddened. The catarrhal inflammation may likewise be a part of a general mucous membrane involvement in the adjacent structures, especially the soft palate, pillars and tonsils. Laryngitis may coexist.

The most common subjective symptom is pain during swallowing, often not definitely localized by the child, but which on examination can be ascribed only to the pharyngeal inflammation. Tenderness of the neck is due to the associated swelling of the regional lymph glands, at the angle of the jaw and back of the sternocleidomastoid muscle.

The vault of the pharynx may be involved, and as a result eustachian tube infection occurs, often followed by otitis media. Pyelitis is not an infrequent complication.

*Treatment.*—As the primary disease is self-limited, the treatment should be directed to the comfort of the child. In infants, cool water and in older children, shaved ice should be given every hour. Fruit juices, soup, milk and cereals are suggested as the principal articles of diet. A hot bath and stimulation of the skin add to the well-being. In case of constipation a laxative should be given daily, some form of magnesia being advisable for its alkaline action upon the urine. Repeated vomiting may be stopped by gastric lavage of sodium bicarbonate solution. Restlessness, fever and pain will be benefited by acetylsalicylic acid, 1 grain at one year of age, 2½ grains at five years and 5 grains at ten years, every four hours.

Acute secondary pharyngitis should have, in addition to the foregoing, whatever specific care is indicated in the primary disease, whether diphtheria, scarlet fever or measles. Removal of adenoids and diseased tonsils should be considered after recovery from the pharyngitis.

**Actinomycosis.**—Actinomycosis of the pharynx and soft palate has been occasionally reported in children. Small yellow nodules are found which on examination reveal the presence of the ray fungus. Secondary infection of the gastro-intestinal tract may occur. Montgomery cured such a case with cauterization and the internal administration of iodid of potassium.

## THE TONSILS

### ACUTE INFLAMMATION OF THE TONSILS

In childhood tonsillitis frequently begins with vomiting and diarrhea and sometimes abdominal pain. Often to the surprise of the parents as well as the physician the cause of the disturbance is found in the pharynx and fauces. Many times I have been told that the child is subject to "bilious" attacks, and when called to attend a typical seizure, find a definite acute tonsillitis. The mother not infrequently remarks that there had been no complaint of "sore-throat." In every case of acute disease of the tonsil the question should be answered as to whether it is one of the following:

Tonsillar diphtheria

Scarlatinal tonsillitis

Vincent's angina

Peritonsillar abscess

Retropharyngeal abscess

Acute follicular tonsillitis (lacunar, cryptic, infective)

**Tonsillar Diphtheria.**—While the tonsils themselves are usually at first alone affected, diphtheria must be considered in a broader sense than that indicated by a tonsillar localization. In children who are negative to the Schick test, a membrane appearing upon the tonsils is probably not due to Klebs-Loeffler infection, but in any case a dirty thick adherent patch larger than the usual cryptic deposit calls for a prompt dose of antitoxin. I have seen a few cases of typical follicular disease develop a definite membrane on the second or third day, not only positive to bacteriological examination, but productive of later diphtheritic palatal paralysis. Diphtheria may be implanted upon many acute inflammations of the tonsils. Zingher has called attention to the secondary invasion of the operative membrane following tonsillectomy, an insidious and difficult complication to diagnose.

The frequent early involvement of the uvula makes easy the diagnosis of diphtheria of the tonsils, but the disease should be recognized and treated when any thick membrane is observed, without waiting for spreading or for confirmation by bacteriological examination.

**Scarlatinal Tonsillitis.**—It is seldom that the tonsils are the only oral structures involved in scarlet fever. Close inspection will usually reveal scarlet inflammation of the mucosa of the fauces, the pharynx, the cheeks, the papillæ of the tongue (strawberry tongue). There are, however, mild cases with little or no apparent sore-throat, and with little fever. The angina of scarlet fever varies from a simple redness and swelling of the tonsils, to a follicular or membranous tonsillitis. Early, it may resemble a simple sore-throat, but after the cutaneous rash disappears, the characteristic scarlet erythema is apt to develop. The membrane is usually thin or transparent, but is not always easy to differentiate from the diphtheritic type. Initial vomiting and the appearance of the rash within twenty-four hours aid in the diagnosis. The positive Dick test on the first day or two of a suspicious throat infection has recently proven of much help.

This type of tonsillitis is seen in epidemics of septic sore-throat, transmitted by milk infections. It is due to the hemolytic streptococcus, and may not be accompanied by scarlet rash. In the light of recent observations it seems that an individual while immune to scarlet rash, may be susceptible to the throat infections. During an epidemic of scarlatina involving the children of one family, the mother, who earlier in life had the disease, developed the typical scarlet angina.

**Vincent's Angina.**—An ulcerative or necrotic type of tonsillitis is now well known, due to infection with the spirillum and fusiform bacillus. From my personal observation, tonsillitis from this cause is rare in children, even in those who have previously had an infection of the gums from the same cause. The disease is characterized by a pseudomembrane covering the ulcerated patches, due to the necrosis of the mucous membrane. The ulceration is sometimes deep and may cause marked loss of tonsil tissue. The membrane is grayish-white, appears in patches, with a surrounding red zone. The microorganisms show the same tendency to invade the deep structures, as found in the jaw and cheek in ulcerative stomatitis and in noma. There is also the same tendency to bleed as is seen in gingivitis. The membrane is in some cases difficult to diagnose from diphtheria. It usually begins on one tonsil but may spread over the fauces.

Symptoms are: Ulceration of the tonsils, with a pseudomembrane, a fetid breath, salivation, an early enlargement of the glands of the neck on the affected side. General symptoms, not always present, are mild fever, chilliness, headache, vomiting, more or less painful deglutition. In some instances the symptoms may be acute and severe. The ulcers may be slow to heal, requiring several weeks or longer.

In many cases antitoxin is given under the assumption that the condition is diphtheria. No benefit will be obtained from it in Vincent's angina. The diagnosis is readily made by a stained smear from the pseudomembranous area. The treatment is specific and promptly curative. Of first importance is the subcutaneous injection of sulpharsphenamin, 0.2 to



0.3 gram for two or three daily doses. Neutral neo-arsphenamin may be used intramuscularly. Potassium chlorate 3 grains by the mouth is to be given between meals. Local treatment is given with the atomizer or by swabbing three times daily, using a solution of neo-arsphenamin in glycerin. Peroxid of hydrogen is a useful mouth wash and gargle to be used alternately to the local application of arsphenamin at four-hour intervals. The ulcers should be dried and touched with tincture of iodine once daily.

In early cases the results are more prompt and the necrosis of little extent. The prognosis is good, and cure results in all cases which have not been neglected.

**Peritonsillar Abscess (Quinsy).**—This is fortunately rare in children, but must be considered as a possibility when abscesses of the fauces and pharynx are to be differentiated. Repeated attacks as seen in adults are in my experience unknown in childhood. Because of the age when it appears it is not to be confused with retropharyngeal abscess of infancy, the late resemblance to which is sometimes marked. In the early stages of both, the location of the swelling is entirely different, that of quinsy in front and external to the tonsil, that of retropharyngeal abscess posteriorly in the wall of the pharynx.

Quinsy is primarily an infection of the tonsillar crypts, chiefly those which open into the supratonsillar fossa. The abscess develops to the outer side of the tonsil and usually points about one-fourth of an inch from the border of the anterior pillar, or near the upper third of the tonsil, occasionally near the posterior pillar.

At first the child complains of pain in the throat but there is no evidence of involvement in the tonsillar region at the early stage of the disease. The symptoms of the well-developed case are, however, dependent upon the marked swelling and abscess formation in the region of the tonsil usually of one side. Peritonsillar tissue and the pillars of the fauces are likewise swollen and edematous. The tonsil is sometimes pushed inward well toward the median line. Much redness and edema are present in all of the structures on one side of the fauces. Fluctuation becomes detectable as the disease progresses. There are constant pain in the throat, difficulty in opening the mouth and in swallowing, and an accumulation or drooling of saliva. The facies is anxious, the breathing is difficult and suffocation seems imminent. There is regurgitation of fluids through the nose. Fever is high in children and the leukocyte count increased.

In from five days to two weeks the abscess empties itself through the anterior pillar or through the tonsil itself and recovery takes place. Surgical drainage of the abscess permits of a much earlier convalescence. The dangers of peritonsillar abscess are slight, but there are few diseases which cause so much discomfort and apprehension. Severe outcome from accident is dreaded but is seldom reported.

Treatment is by early application of cold compresses, cold drinks, and

later by inhalations. Radiant heat and acetylsalicylic acid give relief. The anterior pillar should be anesthetized and dissected from the tonsil, and a blunt dissector carried backward between the capsule and the bed of the tonsil until the pus is reached.

**Retropharyngeal Abscess (Retropharyngeal Lymphadenitis).—**

Retropharyngeal abscess is a disease of the retropharyngeal lymphatic glands, occurring in infancy, and is the result of infection coming from the nose and ear, nearly always from rhinitis, coryza, sinusitis and otitis media. It is less frequently due to contagious disease such as measles or scarlet fever. A recent series of fifty-nine cases in young children was reported in New York City by Sheffield. The cases reported occurred during the presence of an epidemic of influenza. Certain of the children were free of tonsils and adenoids, and the age of most was below three years.

Susceptibility is increased in children who are suffering from nutritional disturbances and syphilis. The acute type occurs almost entirely in infancy and is primary. When the abscess is secondary, it nearly always comes from caries of the vertebra, usually tuberculous. This is a "cold abscess," much slower in course than the acute abscess of infancy. Cold abscess is more apt to appear in children past the age of three years.

The infection is streptococcic or staphylococcic. The pus develops in the cellular tissue behind the posterior wall of the pharynx. Infection travels by way of the lymphatics to one or more of the retropharyngeal or prevertebral glands, situated near the axis, which corresponds with the level of the posterior portion of the tongue. There is no barrier to the downward passage of the pus, so that a fistula may form leading to the lower part of the neck or into the thorax.

Infection may cause only inflammatory changes in the glands but usually leads to abscess formation.

*Symptoms.*—After an illness of five to ten days due to pharyngitis, purulent rhinitis or the ordinary "cold" the infant becomes restless, with fever constantly present and at times high and with periods of chilliness. These are followed by the development of lymphadenitis. One of the most characteristic symptoms then appears, *painful and difficult swallowing*, a condition which increases with the growth of the abscess behind the pharyngeal wall. The child refuses food and may choke on attempts to swallow it. *Dyspnea* is a symptom of nearly as much importance as the difficult swallowing. It is due to local pressure at the site of the abscess, and the amount is due to the size of the obstructive mass. In addition to difficulty, the breathing is noisy because of the rattling of mucus in the pharynx. When one inspects the chest he will notice that there is a substernal tug with each inspiration. Cough, vomiting and regurgitation through the nose may be present.

*The child holds the head markedly retracted* and toward the affected side (wry-neck). This is one of the regular and diagnostic findings. This



position seems to relieve the obstruction, or at least to make the child more comfortable. *The cry of the infant is peculiar* and belongs especially to this affection, being due to an obstructed throat. The characteristic of the cry is that it is short and smothered with a nasal quality. There is some swelling of the neck at the angle of the jaw on the affected side, the submaxillary glands being usually involved, and occasionally the parotid. The neck is

stiff, and attempts at movements cause much pain.



FIG. 9.—CHARACTERISTIC RETRACTION OF HEAD AND GLANDULAR SWELLING OF NECK ON THE AFFECTED SIDE

Inspection of the throat shows that it is red and swollen, and distinctly bulging on one side, in the lateral pharyngeal wall. It is difficult to view the throat in these infants both because of the smallness of the mouth and the pharynx and because of the resistance to examination, the pain caused by stretching the jaws and the presence of much mucus. The examination of the pharynx by the finger is therefore a useful supplement to inspection. In this way *the bulging suppurative mass* may be felt. Bulging is not seen early in the dis-

ease in young infants. It will cover considerable area extending from the back of the pharynx forward on one side and from the posterior nasal space downward below the pharynx.

The disease lasts from one to three weeks. Death may occur from a paroxysm of dyspnea. Asphyxia is usually due to neglect of treatment, by which the obstructive mass is not drained sufficiently early. The abscess seldom ruptures spontaneously. Pus may travel down to the mediastinum. Sepsis, pleurisy and pneumonia may result.

The mortality rate depends almost entirely upon the time of diagnosis and drainage of the abscess. Early evacuation of pus causes prompt recovery. Sheffield reports that all of his forty-five cases recovered, some of them taking two or three months.

*Diagnosis.*—Following an upper respiratory infection, the development

of retropharyngeal abscess in infancy is marked by dysphagia, dyspnea, retraction of the head, peculiar muffled cry, swelling of the affected side externally and bulging internally. Pus will be found in the mass when it is incised.

The differential diagnosis is to be made from peritonsillar abscess, from faucial and laryngeal diphtheria, and the bulbar type of infantile paralysis.

Quinsy is extremely rare in infancy, while this time of life is the period for retropharyngeal abscess. In the former the pus comes from the tonsil in front, in the latter from the deep glands posteriorly. In late diphtheria the throat is very much swollen and it might be extremely difficult to rule out an abscess. The presence of a characteristic diphtheritic membrane will help in the diagnosis. Laryngeal diphtheria is not so easily differentiated, until the bulging retropharyngeal ab-

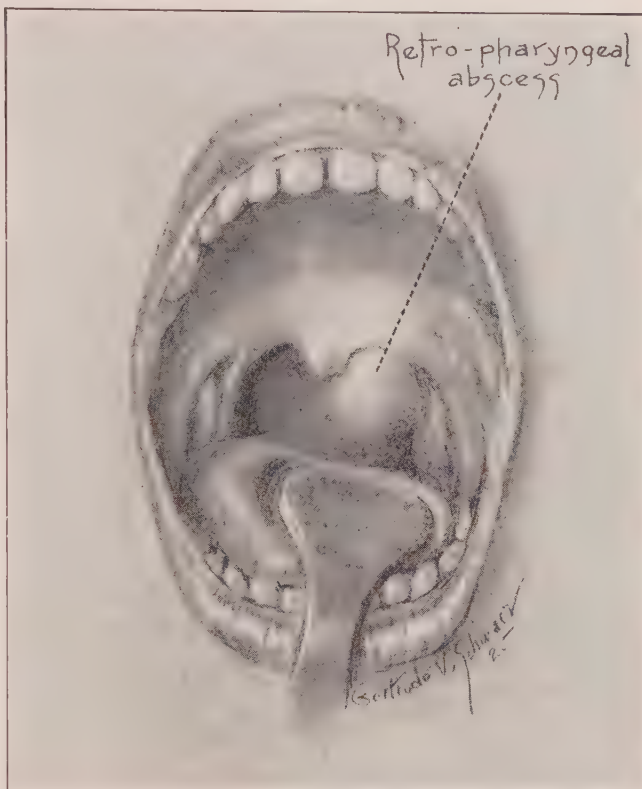


FIG. 10.—DRAWING ILLUSTRATING THE SITE OF THE ABSCESS

cess can be seen or palpated. Infantile paralysis appears more quickly after the initial fever. Paralytic signs in the throat with much collection of mucus will be found in the bulbar type, without any evidences of pharyngeal bulging.

*Treatment.*—Local measures are the early irrigation of the nose and throat with mild antiseptic solutions such as Seiler's or boric acid solution, every three hours. The instillation of organic silver preparation in the nose may be of some benefit. External heat gives comfort to the child. The child should have plenty of rest. General treatment with tonics and nourishment is necessary.

Surgical treatment consists of a wide and deep incision in the presence of an abscess. This is done through the mouth. Visualizing or palpating the area to be incised is not an easy matter, as the jaws are separated with

difficulty. The operation is usually done by the sense of touch, both in locating the point for incision and palpating the floor of the opened abscess. In some cases the glands need additional opening from outside dissection.

Certain difficult cases are inaccessible by the mouth and require a careful external operation with drainage.

**Acute Follicular Tonsillitis.**—The health of the child may be seriously impaired by the occurrence of repeated tonsillitis. When one considers the damage done to the structures of the tonsil by inflammatory attacks, any function which it may have possessed in health is insignificant, and the latent danger of severe infections outweighs all other considerations.

Acute inflammation is due to the lowered resistance of the epithelium lining of the crypts and the invasion of the pathogenic microorganisms. The occlusion of the crypts is responsible for the impairment of the epithelium and for the presence of infectious foci. Toxins liberated by the retained microorganisms damage the lining of the crypts, resulting in entrance of bacteria into the tonsillar tissue and the general circulation. Previous attacks of tonsillitis cause permanent narrowing of the crypts. Food and debris are thereby retained. There is also anatomical interference with the drainage of the follicles in the portion of the tonsil covered by the pillars. Tonsillitis is most often caused by some type of streptococcus, commonly the hemolytic, which may remain in the lacunæ after the attack and cause reinfection whenever the conditions are favorable. The staphylococcus, pneumococcus and other bacteria inhabiting the mouth are responsible for some cases.

Just what effect "catching cold" has in inaugurating acute tonsillitis is doubtful. It probably acts by producing a lowered resistance, but it can at most be only one of the factors which excite inflammation of the tonsil.

**Local Symptoms.**—Both tonsils are usually involved. During the attack the structure is markedly red and swollen. The follicles are filled or covered with a yellowish-white deposit, composed of leukocytes, desquamated epithelial cells and numerous microorganisms. Pus may be seen coming from the openings. In some cases the deposits are more extensive, appearing as patches of pseudomembrane in the nature of fibrinous exudate. It is these larger and pseudomembranous exudates that sometimes make the differentiation from diphtheria difficult.

The inflammation extends to the surrounding mucosa of the pillars and pharynx. The tongue is coated and the back of the mouth often shows an accumulation of mucus. The breath is offensive, and when the swelling of the faucial structures is severe the speech is impaired. There is much pain in swallowing, and the glands at the angle of the jaw are swollen and tender, and cause pain when the head is turned.

**General Symptoms.**—Fever is present in all cases, especially on the first and second days. In young children the temperature reaction is high,



usually around  $103^{\circ}$  F., and in some cases during the height of the inflammatory process up to  $105^{\circ}$  F. There may be chilliness, headache and joint pains. The appetite is absent during the febrile stage, and deglutition is painful because of the inflammatory changes. The urine not infrequently contains albumin and granular casts, as a result of the toxic degeneration of the tubular lining of the kidneys. In all cases the urine should be examined for the presence of blood, casts and pus.

Usually the tonsillitis subsides in from three to five days and the child soon resumes its usual state. But in severe cases and after repeated attacks the general condition may be greatly weakened by the toxemia, the loss of food, and the complications. These may be suppurative cervical adenitis, pyelitis, hemorrhagic nephritis, rheumatic fever, chorea, endocarditis, or anemia. No case of tonsillitis should be dismissed without subsequent general examination at intervals.

*Diagnosis.*—In childhood the question of diphtheria and scarlet fever must be considered in all cases of tonsillitis. The throat during the acute stage should be daily inspected by the physician. Diphtheria should always be borne in mind, and the rash and desquamation of scarlet fever not overlooked. High temperatures are more common in follicular tonsillitis than in diphtheria. The course of tonsillitis is progressively better, that of diphtheria is marked by extension of the membrane. When palatal paralysis occurs it is certain that diphtheria has been present. As above stated, it is better to regard as diphtheria any tonsillitis in which even small patches of membrane occur, and to give antitoxin.

*Treatment.*—The tendency of recent practice is to disregard local treatment during tonsillitis. This has come about because of the recognition of the self-limited nature of the disease. It was formerly the custom to use a local application of tincture of iodine and glycerin, in a 1 : 8 solution. The pain and annoyance caused to the child by any sort of oral therapy make one hesitate to use a method of treatment the value of which seems unproven. However, on the first day of a tonsillitis which is productive of marked swelling and pain, there would seem to be justification for the practice of painting the surface with a strong solution of silver nitrate (50 per cent). Ballenger highly recommends this, care being taken that no excess of the caustic be allowed to drain off upon the surrounding tissues. Only in those children who are easily treated, and in the hands of the expert should such a drastic remedy be attempted.

The treatment should be directed to the comfort of the child. In some it may be possible to keep an ice collar over the swollen submaxillary glands. Plenty of water should be given so as to stimulate the renal excretion, and allay thirst. A laxative of milk of magnesia three times daily aids in the elimination of toxins, and may keep the urine alkaline and prevent pyelitis. Acetyl salicylic acid every four hours relieves pain and reduces fever,



whether it has any prophylactic effect upon cardiac and rheumatic complications.

After caring for a child during severe tonsillitis, I do not feel that I have filled my obligation without urging the removal of the tonsils and the consequent prevention of future attacks.

## THE RÔLE OF LYMPHOID DISEASE IN THE PHARYNX AND FAUCES

Undue hypertrophy of the lymphoid tissue in the pharyngeal wall, the rhinopharynx and the tonsillar fossæ has a direct bearing upon the welfare of the child. Hyperplasia may be the manifestation of a predisposition which some individuals possess, known as a lymphatic diathesis. There is something peculiar to childhood in the tendency of lymphoid structures of the nose and throat to become chronically enlarged. There are various causes for this stimulation of lymphoid growth. Occasionally it is congenital, sometimes it may be due to overfeeding during infancy and early childhood (Czerny), but in most cases the increase is due to the effect of repeated infections in the upper respiratory tract.

Enlarged tonsils are seldom present in infants, but appear in the second year and later. I can recall very few infants with large tonsils, but many with definite enlargement of the adenoids. The youngest child I have known to undergo a tonsillectomy was six months old. This was a Negro who had marked obstruction from the excessive size of the tonsils and adenoids. The operation was attended by no untoward results, and the removal greatly benefited the child.

**Anatomical Changes in the Tonsil.**—The size of the tonsil is not always easily determined from inspection through the mouth. Much of it is hidden in the tonsillar fossa. One often sees tonsils which meet in the middle line. When the tongue is depressed and gagging is caused by the application of the tongue spatula, the size of the tonsils is more readily observed. The buried or submerged tonsil is hidden behind the pillar, but may be considerably larger than its appearance indicates. Ballenger recommends palpation with the finger as a means of determining the size.

In children all of the structural elements of the hypertrophied tonsil are hyperplastic. The growth is softer and smoother than found in older individuals. In the submerged tonsil there have been fibroid changes which caused the shrinking. It is probable that in most hypertrophied tonsils the crypts are obstructed or unhealthy; in either event they are susceptible to microbic invasion.

Clinically the principal anatomical factor is the lacuna or crypt, which is an epithelial ingrowth into the connective tissue, forming a tubule which penetrates the tonsil clear through to the capsule. These crypts easily fill with food débris and microorganisms and are readily attacked by inflamma-

tory processes when the mouths of the crypts become closed and cannot drain. Organisms are taken up by the epithelium and the result is local or general disease.

Normally the tonsil is compressed by the pharyngeal and palatine muscles which may force food either into or out of the crypts. Healthy epithelium of the crypts counteracts infection but a loss of tissue tone favors it. The tonsils when diseased must be regarded as portals of infection. There has been much discussion about their function. Probably when healthy they have a protective action, but in any case the anatomy of the crypts is such that they retain and favor the growth of organisms. If the body resistance or the integrity of the epithelium becomes lowered the deeper structures become invaded.

Lymphatic vessels originate in the tonsils and communicate with the deep lymphatic glands of the neck, so that infection can be carried from the tonsil through the lymph channels to the thoracic duct, the chest or any portion of the body. If the tonsil becomes diseased the disturbance may remain local or it may produce an insidious and obscure general disturbance.

Repeated attacks of acute tonsillitis should be regarded as evidence of residual infection. Chronic infection of the tonsil may result in hypertrophy, but it is well known that certain small and even buried tonsils may contain foci of pathogenic microorganisms. In any event the tonsillar disease is accompanied by infiltration and swelling of the cervical lymph-nodes which are often visibly enlarged and not infrequently tender to the touch. Especially in the thin child can the hyperplastic glands be seen, when the chin is turned sharply to one side. The submaxillary gland is conspicuous in the triangular space below the horizontal ramus of the jaw. The superficial posterior group of glands when hyperplastic may also be readily seen.

The general health of the child usually suffers. The most serious result is a systemic infection manifested by more or less persistent fever, rheumatic pains, endocarditis, chorea and nephritis. Tuberculosis may be carried from infected tonsils to the lungs or be disseminated to the abdominal and mesenteric lymph-nodes. Tubercle bacilli, when present in the tonsils, may cause local disease therein and involvement of the regional lymphatic glands.

The effect of lymphoid disease is seen in the digestion and nutrition. Obstruction interferes with the ventilation and oxidation in the lungs, as a result of which the body tone and general functions may be disturbed. Affected children do not sleep well at night and are restless during the day. The appetite is greatly impaired and vomiting may result on slight provocation. It is only in the children who have a lymphatic constitution that there is marked overweight. Most children suffer from undernutrition, not only because of lack of appetite, but probably also because of the insufficient utilization of food dependent upon atony of the secretory and motor apparatus of the gastro-intestinal tract. If one wishes to learn the effect

of excessive lymphatic hypertrophy upon the health of children he needs only to observe the striking results often promptly manifested after the removal of obstructive tonsils and adenoids. Associated infection and the tendency to acute inflammatory exacerbations are eradicated by removal of the hyperplastic tissue.

*Removal of the tonsils is to be urged* when there has been persistent local infection therein, when they have suffered two or more attacks of acute inflammation, or when they are greatly hypertrophied so that they nearly meet and cause marked obstruction. Repeated attacks of pharyngitis should cause suspicion of tonsillar infection. There is usually hyperplasia of the lymphoid tissue of the rhinopharynx which adds to the infectious area and to the obstruction. When in addition to these manifestations of lymphatic disease there are such secondary effects as hyperplasia of the regional glands, impairment of appetite and nutrition, or the occurrence of systemic infections, the earliest opportunity should be taken for tonsillectomy. In a large group of children whose tonsils had been removed the nutrition was found to become much better than in a similar number who still possessed tonsils.

The capsule should be removed with the tonsil, so as to include all source of infection. Roentgen-ray treatment is as yet only to be considered when the child is a poor operative risk.

**Adenoid Vegetations.**—Excessive growth of lymphoid tissue in the vault of the pharynx has a direct bearing upon the digestion and nutrition of the child. The primary results are retention of infectious microorganisms and lowered resistance to infection. Such children have almost constant "colds" and disturbed nasal ventilation. The adenoid mass may cover the vault and the eustachian orifice, as a result of which deafness and repeated ear infection may be caused. When the mass extends downward over the angle of the pharynx, it may be visible by direct inspection of the throat. It is sometimes the cause of repeated pharyngitis. As a result of the numerous infections caused by this unhealthy tissue, the child frequently has either a steady loss of appetite or recurring periods when little food is taken. Restlessness and impaired sleep add to the nutritional losses.

Secondary changes in the upper jaw cause interference with proper mastication and lead to further digestive disturbance. The mouth must be kept open in order to breathe, as a result of which the oral mucosa is dry, the food insufficiently moistened, and decay of the teeth favored by the deficiency in saliva and the accumulation of débris.

When the mouth is inspected the roof is found narrow or high. Normal occlusion of the teeth is impossible due to irregular placing. The upper jaw projects far beyond the lower teeth, so that chewing is greatly interfered with. Habitual thumb-sucking may still further push the upper incisors forward. Unless corrected by orthodontia, the deformity lasts for life.

The end results of persistent adenoid infection and obstruction are seen in the ill health and improper development of the child. There is always a tendency to croup and other respiratory infections, especially asthmatic bronchitis. The sleep is restless; the child is languid and anemia nearly always develops. In the opinion of most observers the obstruction is the most frequent cause of chest deformity, particularly pigeon-breast. Flabbiness of the muscles and underweight commonly result.

A pathological state of such importance calls for radical interference. However much opinion may vary as regards the indication for tonsil removal, there can be no argument advanced against adenoidectomy. The writer has come to the conclusion that no arbitrary rule should be followed as to the age when adenoids should be removed. It is indicated when obstruction or toxic symptoms have developed, even as early as one year of age. Many children will be spared otitis media, sinus infection, nutritional disturbances, and above all, complications during measles and scarlet fever if the nose and pharynx are put in healthy condition in the first years of life. The argument against early operation has been the possibility of return of the adenoids. Such a recurrence does sometimes happen, but the child is given a period of freedom from the symptoms. The untreated case shows no tendency to spontaneous recovery, and during the course pronounced disturbances may become intolerable. Objections to operation based entirely upon the age of the child are usually unfounded.

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## CHAPTER V

### THE SALIVARY GLANDS

**Anatomical and Functional Consideration.**—The salivary glands are present at birth, but are relatively inactive and unimportant during the early period of infancy. Because of the slight amount of secretion, the mouth of the newly born is dry and the tongue coated. During nursing, the saliva is mixed with the milk in the mouth, but because of the liquid nature of the food less secretion is excited. Considerable amount, however, is secreted during infancy, estimated by some observers as representing 10 to 20 per cent of the stomach contents. The saliva has little if any function in facilitating swallowing at the early time of life, for lubrication of the mouth is not needed in the process of deglutition until dry feeding is begun. The normal saliva probably has some inhibitory effect upon the attack of the mucous structures by the oral bacteria. Activity of the glands increases as teething approaches, becoming markedly stimulated by the feeding of substances which require mastication and moistening.

The saliva contains ptyalin even during early infancy, and has some slight amylolytic action. At ten months of age the salivary power of digesting starch is twice that at birth, and soon becomes as great as at any time in childhood. The nursing infant has little need for a starch-digesting ferment, but can well handle amylaceous food when such is added to its dietary. The saliva becomes acid after remaining in the mouth for some time. When freshly secreted, the saliva is usually faintly alkaline, but is sometimes neutral or slightly acid. By the time adult life is reached the reaction is definitely acid. There is probably little digestion of starch until the food and saliva reach the stomach and duodenum.

The size and weight of the glands are proportionate to those of adults. In the newly born the parotid gland, while larger than the submaxillary and sublingual, does not show the marked preponderance in size found in later infancy. It grows faster than the other glands. All the salivary organs increase rapidly in weight, being three times as heavy at six months and five times at two years of age (Scammon). The parotid after infancy outgrows its site in the space between the ear and the masseter muscle and spreads forward over that muscle. It thus assumes the situation that is found in adult life. The microscopical structure becomes mature by the time the child is two years old.

Important anatomical landmarks should be remembered by the physician in the diagnosis of affections of the glands, and by the surgeon who is called

upon to operate upon them. The parotids lie close to the wall of the pharynx, and intimately related to important vessels and nerves. The facial nerve lies in the superficial plane of the glands, while in the deep relations are found the carotid arteries, the internal jugular vein, the *vagus* and glossopharyngeal nerves. Steno's duct has its orifice in the cheek opposite the second upper molar tooth. In epidemic and suppurative parotitis the mouth of the duct shows redness and swelling.

The submaxillary gland lies in the submaxillary triangle just anterior to the angle of the jaw, below and to the inner surface of the mandible. It is palpable only when enlarged. Wharton's duct empties into the mouth by a papilla on each side of the frenum of the tongue.

The sublingual glands are situated in the anterior portion of the space under the tongue, and may be seen normally as elevations covered by mucous membrane on the floor of the mouth, at either side of the origin of the frenum. They have numerous ducts opening in the floor of the mouth, and also into Wharton's duct.

**Disturbances of Salivary Secretion.**—The quantity of saliva is apparently, but not actually, increased when certain diseased conditions are present in the throat and mouth, such as paralysis from diphtheria, bulbar poliomyelitis, facial nerve trauma, and in severe angina, Vincent's disease or peritonsillar abscess. This is due to paralytic or inflammatory interference with swallowing. Ptyalism is an excessive secretion of saliva, a normal process when occurring in infants while cutting teeth. Mentally backward children frequently have persistent drooling. There is probably an associated disturbance in deglutition in some feeble-minded children. The manifestation of drooling is a common one in spastic cerebral diplegia (Little's disease). The most common diseased conditions producing salivation are the various types of stomatitis. Mercurial salivation may occur in childhood, but is now seen infrequently. That mercury will produce salivation in every individual, if given in large enough doses, is well known.

The secretion of saliva is decreased during febrile diseases, because of the lessened stimulation from poor appetite and the small amount of dry food, but also by the desiccation of the body tissues. Common illustrations of this are found during ileocolitis, typhoid fever, and pneumonia.

**Abnormal Development.**—The most common tumor of the parotid is hemangioma. This vascular growth is undoubtedly congenital though not always characteristic until a few weeks or months after birth. Fullness of the parotid region is usually noticed at once, and soon a bluish mass of coiled blood-vessels may be seen through the skin. While the skin is not affected, the growth much resembles nevus. The tumor grows rapidly. It is soft and enlarges with crying. The occurrence of hemangioma in the parotid is relatively common. The writer has seen three such tumors of the parotid; the surgeon or dermatologist will have referred to him a not inconsiderable number.

Hemangioma of the parotid is benign, but as stated by von Reuss is not harmless. If left alone it always causes death. The size rapidly increases until the cheek and neck on one or both sides may be involved. By pressure upon the larynx the tumor may produce suffocation.

The treatment is early removal of the growth while it is still small. Injury to the facial nerve and hemorrhage are serious factors in the surgery of this type of tumor. The complete resection of the growth results in cure.

The parotid may be poorly developed or in rare instances missing. A father and his child were found to have a complete absence of the salivary glands (Ramsey), the symptoms of which were the dryness of the mouth and the early decay of the teeth.

**Salivary Cyst.**—The most common type of cyst affects the ducts of the sublingual gland. It is often known as ranula because of the frog-like appearance when the growth is bilateral. The French use a term with a similar meaning—*grenouillette*. There is a cystic enlargement underneath the tongue resulting from obstruction of the duct. It is not uncommon in children. The growth is bluish in color, soft and fluctuating. Back of the obstruction there is retention of saliva and mucus. It may be on one side alone or extend to the opposite side of the frenum. The cyst is unilocular. The size increases slowly, but may develop to the extent of interfering with nursing or swallowing, markedly reducing the nutrition and causing underdevelopment of the teeth or jaw.

In some cases the origin is undoubtedly due to embryonic maldevelopment, and in rare instances there may be an atresia of the canal. In this event the ranula will be noticeable at birth. In others the cyst follows a chronic inflammation of the duct with sclerosis of the canal and a dilatation behind the occluded portion.

Salivary cysts of the parotid and submaxillary are much rarer than ranula. In a case known to the writer a large multilocular salivary cyst of the parotid interfered with the delivery of the fetal head at birth. The growth was successfully removed on the sixth day of life, with no recurrence or untoward effect.

Lymphangioma may affect any or all of the salivary glands. The growth does not grow rapidly or threaten the life of the child as does hemangioma.

**Mixed Tumors of the Parotid.**—A variety of growth, designated "mixed tumor," affects the salivary glands, but is only rarely reported during childhood. It is said to be more often found in the parotid than in the submaxillary gland. Such a tumor is encapsulated, lobular, and may contain numerous types of tissue, connective or fibrous, cartilaginous or mucous. The growth will be hard or soft, depending upon the kind of tissue which chiefly makes up the content. The tumor is easily movable, is

irregular in shape due to nodules. The course is benign, but may in some cases become malignant.

A case reported by Wood was in a seven-months-old child. The tumor appeared at birth, was small at first, and then rapidly developed.

Removal becomes necessary because of the large size and discomfort.

Sarcoma, adenoma and lipoma of the parotid have been reported in children but must be regarded as extremely rare.

#### **Mikulicz's Disease.—**

This is a clinical syndrome, characterized by chronic, painless, symmetrical enlargement of the salivary as well as the lacrimal glands. While occurring mostly in later life, the collected cases will be found to include a number of children. It can be of congenital or familial causation. Von Reuss reported a five-and-one-half-year-old girl who for several years had a slowly developing swelling of the parotids. The size would be augmented during acute nose and throat infections, at which time fever and pain were present. At other times the child was free



FIG. II.—LARGE MULTILOCULAR SALIVARY CYST OF THE PAROTID IN THE NEWLY BORN INFANT

of symptoms, but small discrete nodular growths could always be felt. The same writer has published the reports of seven cases in children.

The disease begins with a swelling of the lacrimal glands, and later the salivary glands and the mucous and secondary glands in the hard palate become involved. The blood-picture may be normal until several years after the beginning of the disease. No fever or marked discomfort is caused, but there are dryness of the mouth and embarrassment to chewing and swallowing. The eyelids and eyeballs may be invaded.

Hase found that the minute changes were in some cases a hyperplasia of the lymphatic tissue and in others granulation tissue of a tuberculous nature. The increase in size of the glands is not due to any hypertrophy of the salivary structures but of the lymphatic tissue in the glands. The



cause of some cases has been ascribed to the localization of infection from the blood. A connection is found with pseudoleukemia in a portion of the cases, when the lymphatic glands will also be enlarged, especially in the conjunctiva, orbits, neck or jaws.

Another class of cases is associated with leukemia, several children with this disease having been reported. The writer saw both parotids involved in a young child during the last stages of leukemia. Leukemia as a cause of the syndrome of lacrimal and salivary glandular enlargement was known before Mikulicz described his disease in 1888. However many of the cases described as Mikulicz's disease do not have an involvement of the lymphatic and blood-making organs, some involving the salivary glands only.

A case reported by Howard was in a six-year-old girl. The lacrimal, parotid, submaxillary and sublingual glands were enlarged, the eyelids were swollen, exophthalmos and a general lymphatic enlargement were present. Both the lymphatic and salivary glands as well as the testes may be involved in the same child.

The glandular enlargement in Mikulicz's syndrome is best treated by the roentgen ray.

**Epidemic Parotitis.**—Epidemic infection (mumps) of the salivary glands is a primary affection, occurring chiefly in the parotids, but coincidentally may affect the submaxillary and sublingual glands. Both sides are usually involved. The writer has seen the submaxillary glands alone affected. The sudden bilateral swelling, not limited to the gland alone, the history of contagion, the self-limited nature and rapid subsidence of the swelling, make the diagnosis certain. Mumps is to be expected at some time during childhood, and is far more common than all other enlargements of the parotid. In doubtful cases it may be hard to state definitely that they are epidemic. It is well to watch for the disease in other members of the household who have been in contact with the case.

**Parotitis (Non-epidemic).**—Simple inflammation of the submaxillary glands may occur with the eruption of teeth. I have seen this happen during the cutting of the six-year molars. Not infrequently the sublingual glands become swollen during the eruption of the incisor teeth after which the inflammation subsides rapidly. Suppuration does not take place.

Acute infection is not common. The parotid gland is more apt to be involved. Suppuration usually accompanies the inflammation. The disease is secondary and appears in the course of septicemia or from neighboring diseases of the ear or bones. Infection takes place through the blood stream, by way of the lymphatics, but most usually through the salivary ducts. Osler mentions the occurrence of salivary-gland infection during typhoid, dysentery, pneumonia, pyelitis, and the eruptive diseases. In the second week of acute ileocolitis a child at the University of Kansas Hospital developed swelling of one parotid and a few days later the opposite gland. Pus drained from both Steno's ducts for two days after which all symptoms

disappeared. No treatment was needed. The parotid glands are occasionally infected in premature or marantic infants, or in children exhausted by long illness. In the newly born an ascending infection may occur either through the ducts or as a result of inflammation caused by their injury from forceps. Although rare in childhood, purulent disease is relatively more common in the early weeks of infancy. The infective agent is usually the streptococcus, staphylococcus, rarely the fungus found in thrush.

As a result of infection, the glands become enlarged, noticeably in the neighborhood of the ascending ramus of the jaw. Both sides are sometimes involved. The overlying skin is found to be reddened, and becomes adherent to the tumor mass. Pain, tenderness and edema are present, but there is not often fluctuation. Pus may be squeezed from the mouth of Steno's duct, which is swollen and red. Fever, poor appetite and loss of weight found in the child who has been previously well can be properly ascribed to swelling of the glands, but in the cachectic febrile child, it is seldom that any general symptoms can be assigned to salivary adenitis alone. The local inflammation and increase in size give the clue to the diagnosis.

The glands are tender to the touch. In some cases the child may have a normal appetite, in other instances he may be too sick to take food. Spread of the pus to adjacent regions of the ear or into the neck and mediastinum is imminent in severe cases, while general absorption, sepsis and exhaustion may occur. Involvement of the facial nerve by paralysis is easily possible. Intestinal infection from swallowed microorganisms is rare.

Parotid infections tend to suppurate; some recover spontaneously, while others if not treated surgically go on to the death of the child.

*Diagnosis.*—Pyogenic infection of the parotid gland usually causes high fever and marked swelling of the cheek in front of the ear. Non-epidemic swelling is circumscribed and firmer. It is usually unilateral. It causes more pain than in mumps, the skin is red and tight over the parotid. A primary disease such as purulent otitis media, typhoid, or smallpox may be present. The infection of the gland may occur as a metastatic process from infections elsewhere in the body, and especially from the mouth. It results rarely from abscess of the mother's nipples.

Fluctuation is not conclusive for the presence of pus, as the fluid content may be due to retained saliva. Edema is usually present in abscess. Of marked value is a high leukocyte count. Pus may be seen draining from the reddened mouth of Steno's duct.

In epidemic parotitis the swelling is usually bilateral with a history of contagion. Redness of the skin is absent, the swelling not confined to the gland. The disease is self-limited. There is a normal blood-picture. Metastasis to the testes, ovaries or breast may occur.

One or both parotids may be enlarged from saturation with iodine medication. The glands become noticeably increased in size in some cases of primary anemia.

*Treatment.*—Local applications of heat are indicated for the relief of pain. Gentle massage over the gland and duct is recommended. Early opening of the abscess should be made, with care that the facial nerve be not injured. The results of early drainage are favorable in the vigorous child.

**Submaxillary Inflammation.**—The submaxillary glands only may be affected, or, in the course of a general involvement, all the salivary glands. The infection occurs in the same manner as the parotids, and the duct of Wharton is filled with microorganisms, leukocytes and epithelial cells. The glandular tissue is surrounded by an inflamed mass of connective tissue, which becomes edematous and purulent. The gland is hard, hot to the touch and painful. Both sides may be involved. Pressure will result in the escape of pus from the mouth of the duct. The swollen gland bulges, both on the skin and mucous surfaces. It compresses the tongue and interferes with swallowing.

The child is feverish, sweats easily and loses weight to a degree that marasmus may develop, and death ensue.

The diagnosis must be made from epidemic mumps affecting these glands. Disease of the submaxillary salivary glands should not be confused with a cervical lymphadenitis.

Hot applications may give relief, but the treatment is by early drainage of the abscess.

**Sublingual Inflammation.**—Rarely one encounters a suppurative disease of the sublingual glands. In the experience of the writer, inflammation of these glands has healed spontaneously, and in the newly born may be a simple primary inflammation. But suppuration may occur and surgical treatment be necessary. The outcome is more favorable in this location.

**Salivary Stones.**—The most common illustration of lithiasis is seen when the salts, chiefly calcium, from the saliva frequently become deposited upon the teeth near the gums. Tartar favors absorption of the affected portions of the teeth, and also predisposes to inflammatory disease of the gums. Stones are occasionally formed in the salivary ducts in children, and have been reported in early infancy. Stone in the gland itself is rare. The obstruction of the sublingual ducts causes sufficient swelling to interfere with nursing. Von Reuss mentions a case in which the stone, protruding from the sublingual duct, was removed by first pressing on the duct and then grasping it with forceps. Calculus originates by precipitation of lime from the saliva, caused by local bacterial disease of the ducts, with retardation in the flow and the thickening of the secretion. The stone is usually about the size of a pea.

The obstruction may cause recurring pain and an intermittent swelling. The sight of food or its presence in the mouth will cause an increase in the salivary secretion, which accumulates in the duct back of the obstruction. This distention produces pain (salivary colic). As the duct dilates, saliva

will escape past the stone, relieving the pain and swelling. In such a case known to the writer, a boy was excused from school every time the parotid became enlarged, the condition being regarded as mumps.

Diagnosis of stone is easy only when the hard mass may be palpated or visualized by the radiogram.

The treatment is removal by operation.

**Syphilis.**—Syphilis may affect the salivary glands. The spirochete has been found in the parotid gland of the newly born infant. The infection may attack the glandular tissue at any age in life, and in any of its forms.

**Lymphatic Enlargement.**—Enlargement of the parotid may occur from infection of the small lymphatic nodes within it. This may be caused by tuberculosis, although such infection is rare in the salivary glands. The enlargement may accompany generalized cervical lymphadenitis.

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## CHAPTER VI

### THE TEETH

**Dentition** is the development and eruption of the teeth. It is probable that to many physicians the term implies only that process which is concerned with the swelling of the gums and the eruption of the teeth into the mouth. The significance of dentition is much broader than usually considered, and concerns the normal embryology, the normal development in fetal and postnatal life, hereditary and nutritional factors, and the so-called disturbances at the time of eruption. The functional, clinical and pathological characteristics of the teeth, as found in childhood, are of great importance, for they directly influence health. It is unnecessary here to review the normal embryology of tooth formation.<sup>2</sup>



FIG. 12.—THE DECIDUOUS TEETH AT EIGHT MONTHS<sup>1</sup>

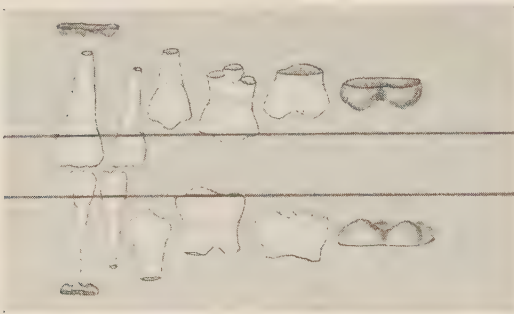


FIG. 13.—THE DECIDUOUS TEETH AT FOURTEEN MONTHS

### THE DECIDUOUS TEETH

**Eruption.**—There is some variation in the eruption of the deciduous teeth, anomalies in the time and order of their appearance being not infrequent. One is puzzled when attempting to explain the exceptions to the rule when certain normal well-

nourished breast infants fail to cut any teeth before ten or twelve months of age, while some infants markedly undernourished and even with rickets,

<sup>1</sup> These drawings of the teeth in infancy (Figs. 12-15) and childhood (Figs. 17-22) are reprinted through the courtesy of William J. Brady, D.D.S., Kansas City, Missouri, from his monograph, *Development of the Teeth*.

<sup>2</sup> A textbook on anatomy and physiology should be consulted by those who are especially interested in the embryology and anatomy of the teeth. Excellent articles are also found in *Feldman's Physiology of Childhood*, in the *American Journal of Diseases of Children*, 1924, 28: 651, by Arthur D. Black, and 1924, 27: 219, by Donald C. Mebane; and the Chart of Development, *Eruption and Absorption of the Teeth*, by Wm. J. Brady, D.D.S., Kansas City, 1925.

get their teeth at the usual time. Cretins are slow in showing teeth. One will occasionally see an infant with the first molars and with only two or even with no incisors. A certain amount of irregularity in the order of their appearance has no significance.

The number of teeth present at certain periods in infancy is easily remembered by the following formula which I have been in the habit of reciting to students and mothers. It should be considered as only approximate and to represent the average rather than the extremes:

## ERUPTION OF DECIDUOUS TEETH

4 teeth are present by the 8th month	{ 2 lower incisors 2 upper incisors
8 teeth are present by the 12th month	{ 4 upper incisors 4 lower incisors
12 teeth are present by the 16th month	{ 4 upper incisors and 2 molars 4 lower incisors and 2 molars
16 teeth are present by the 20th month	{ 4 upper incisors, 2 molars, 2 canines 4 lower incisors, 2 molars, 2 canines
20 teeth are present by the 30th month	{ 4 upper incisors, 4 molars, 2 canines 4 lower incisors, 4 molars, 2 canines

The following diagram also shows the time and order of appearance:

6th to 7th month	$\frac{\text{---} \quad \text{i} \quad \text{i} \quad \text{---}}{2} = 2$
8th to 10th month	$\frac{\text{---} \quad \text{i} \quad \text{i} \quad \text{i} \quad \text{i} \quad \text{---}}{2} = 6$
11th to 16th month	$\frac{\text{---} \quad \text{m} \quad \text{i} \quad \text{i} \quad \text{i} \quad \text{i} \quad \text{m} \quad \text{---}}{6} = 12$
17th to 20th month	$\frac{\text{---} \quad \text{m} \quad \text{c} \quad \text{i} \quad \text{i} \quad \text{i} \quad \text{i} \quad \text{c} \quad \text{m} \quad \text{---}}{8} = 16$
24th to 30th month	$\frac{\text{---} \quad \text{m} \quad \text{m} \quad \text{c} \quad \text{i} \quad \text{i} \quad \text{i} \quad \text{i} \quad \text{c} \quad \text{m} \quad \text{m} \quad \text{---}}{10} = 20$
$\frac{\text{---} \quad \text{(i = incisor)} \quad \text{m = molar} \quad \text{c = canine)} \quad \text{---}}{10}$	

**Loss of Teeth.**—The deciduous teeth are lost by caries, infection or physiologically by atrophy and absorption of their roots caused from pressure of the permanent underlying teeth. It should be remembered therefore that the tooth at the normal time of shedding has lost its root and is easily extracted. The teeth become loose and may spontaneously fall out or be pulled out by the fingers. Root growth and absorption in the deciduous tooth are interdependent.

**Disturbances during Eruption of the Teeth.**—In the eruption of the deciduous teeth the lower incisors usually appear without discomfort to the infant. The gums of the upper incisors are usually somewhat swollen,

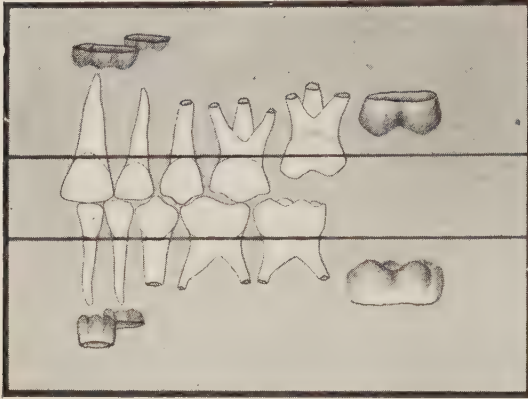


FIG. 14.—THE DECIDUOUS TEETH AT EIGHTEEN MONTHS

tender and more difficult of penetration by the teeth. The deciduous molars likewise may cause considerable discomfort to the child. The canine teeth give less disturbance than the molars.

Kirk thinks that the pain during cutting of teeth is due to pressure of the gum upon the tooth rather than the underlying tooth causing pain in the gum.

It is an unfortunate mistake to assign symptoms or reflex disturbances to perfectly normal gums which have no evidence of distention.

When the gums are red, swollen, tense, edematous or dry, and when the underlying teeth are evidently exerting marked pressure, the child is undoubtedly suffering irritation and discomfort. Even in healthy infants markedly inflamed gums during eruption of teeth may cause digestive disturbance with vomiting and undigested stools. At such a time the child is restless at night, and irritable in the day. In the neuropathic child there may be screaming, convulsions, slight fever, and rarely the appearance of strabismus. One frequently sees swollen submental glands, which disappear after the teeth are completely erupted. Facial eczema may also develop or an existing rash may show marked exacerbations.

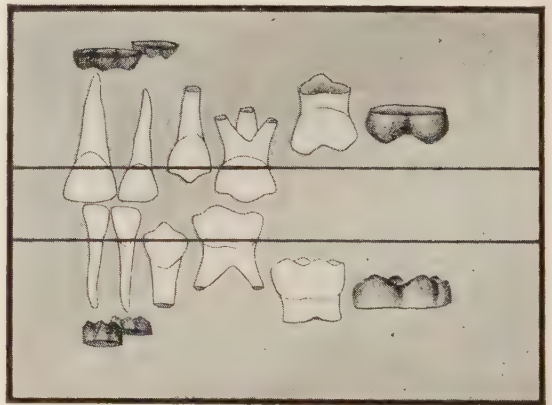


FIG. 15.—THE DECIDUOUS TEETH AT THIRTY MONTHS

Teething is popularly assigned as the cause of many disturbances during infancy. It takes its place along with worms and a long prepuce as a symptom-complex of importance to the laity. Only after a careful general examination and the exclusion of all other causes is the physician warranted in assuming that illness in the child is due to eruption of teeth. I would

suggest that "dentition gingivitis" is a more appropriate term for inflammation of the gums during dental eruption. Teething is usually a normal physiological process without any untoward symptoms or pathological significance.

**Abnormalities.**—Two lower central incisors are present at birth in one of 15,000 infants. Such premature teeth are apt to crumble early. Other abnormalities will occasionally be seen, such as the absence of one or more incisors, the presence of two canines between the lateral incisor and the molar, the occurrence of a double incisor.

Imperfect teeth are due to malformation during intra-uterine life. Occasionally one will see the primary teeth honeycombed with black porous openings. In other cases the teeth will be yellowish, crumbling, and the enamel destroyed. Pitting and horizontal lines date from the time during calcification when the child suffered a nutritional disturbance. Hypoplasia or atrophy of the teeth is not rare.

Craig reported a permanent green staining of the deciduous teeth in a child who was born jaundiced and who remained so for seven months. Such teeth are green when they are erupted.

Syphilis does not affect the temporary teeth. Some observers, however, have ascribed a variety of peg-shaped incisors to such an infection. This conception of the possibility of syphilitic changes in the temporary teeth is contrary to the usual opinion, and should not be accepted until there is more convincing proof. First teeth are not affected because of the fact that severe syphilis at the fetal age when these teeth are developing (four and one-half months) will cause death of the fetus (Jeans).

One frequently sees the spaces between the deciduous teeth nearly as wide as the teeth themselves. This spacing is due to the narrowness of the primary teeth and to the growth of the jaw in accommodating the broad underlying permanent teeth.

**Diseases of the Temporary Teeth.**—There is a marked difference in the vigor of the teeth in early childhood. The primary set is often perfect in spite of lack of care, and at times in the presence of poor general nutrition.

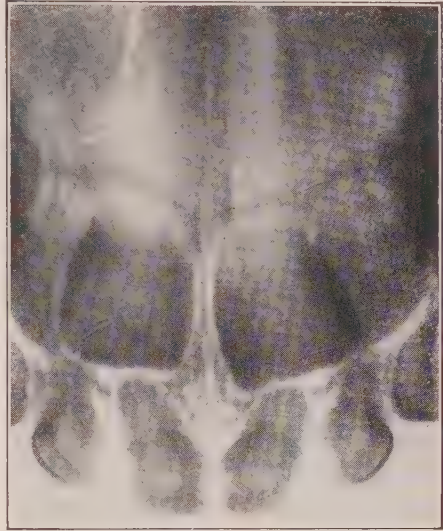


FIG. 16.—RADIOGRAM OF UPPER JAW IN YOUNG CHILD

The deciduous set in the lower row shows physiological absorption taking place. Note the wide spacing between these temporary teeth which provides jaw room for the large permanent incisors just above them and which have not yet become erupted.



These factors however are more apt to result in damage to the dental structures. Normal conditions of the fetus are necessary for the development of the dental buds and the laying down of the minerals. Acute illness in infancy adds to the nutritional disturbance of the teeth. Rickets retards the eruption and calcification. Hypoplasia is due to congenital or infantile lack of retention of lime salts.

The most common disturbance seen in early life is caries of the crown or proximal edges, areas of decay usually involving adjacent sides of the teeth. The crown rapidly crumbles. Multiple caries is usual, and in severe cases there may be no tooth unaffected.

At the same time infections are often present, pus oozing from around the tooth, or from fistulous openings through the gums. Extensive caries is due to the neglect, which is of common occurrence in the children of this age.

The health of the child should be considered above all other questions in the consideration of the teeth. Cavities should be filled with a permanent material while they are small. Badly decayed teeth should be extracted so as to prevent changes in the permanent set. Early decay should be prevented or checked so that there may be no resulting abscess affecting the growing roots of the temporary or permanent teeth. Infection in the early years of life interferes with the complete development of the roots. Abscessed teeth should be extracted, as treatment of such is unwise before the root is complete. Abscess formation causes a delay in the absorption of the deciduous roots. The underlying permanent tooth may thereby be erupted before the deciduous tooth is out of the way.

The persistence in bottle feeding and liquid foods is injurious. Food which requires chewing is advisable, because of the need for exercise by the jaws and teeth.

Calcium and phosphorus, the mineral constituents of teeth, should be generously supplied in the child's diet. Vitamins help in supplying needed minerals. For this reason fruit juices and vegetables should be given during the later months of the first year and throughout childhood. American children as a rule are fed sparingly on whole grains and vegetables chiefly because they do not care for them. Cross reported the examination of 176 newly arrived immigrant children who had never used a tooth brush. Caries was absent in 30 per cent, two small cavities were found in 30 per cent, and very few pronounced cavities in the remainder. Cohen showed by comparison the same number of children, native to America, not selected, in which there were few perfect mouths, and 80 per cent needed dental care. The lesson from this is in the value of the simpler, coarser diet.

A disease which causes loosening and loss of the teeth has been recently described under the names of acrodynia, pink disease, and erythredema. The teeth are not involved in all cases unless the disease is severe. I have seen the dental manifestations in one case, an infant of one year of age,

who lost all of her eight deciduous teeth and two of the underlying incisors during the illness.

## THE PERMANENT TEETH IN CHILDHOOD

### ERUPTION AND NUMBER OF TEETH

First (6th-year molars).....	4 at 5½ to 6 years
Incisors (central) .....	4 at 7 years
(lateral) .....	4 at 8 years
Bicuspid (premolar).....	8 at 9 to 10 years
Canines .....	4 at 12 years
Second (twelfth-year) molars.....	4 at 12 to 15 years
<hr/>	
TOTAL .....	28

The first permanent teeth (sixth-year molars) usually appear by the sixth birthday. The eruption of these teeth may be heralded by the swelling of the submaxillary glands. It is not unusual however for the presence of these molars to be unnoticed until caries or toothache calls attention to them. They are frequently found badly diseased within a few years of their appearance, and are sometimes lost before adult age is reached, thus leaving a vacancy in the jaw for the remainder of life.

The two deciduous molars are replaced by the bicuspid (premolars). The second molars appear about the age of puberty.

Any one of the permanent teeth may be absent, the lateral incisors of the upper jaw more frequently.

In three thousand children of school age, Lyons and Mebane found considerable irregularity in the time and order of the eruption. The sixth year molars may be present at five or not until nearly eight years. All the twenty-eight permanent teeth may be in place by ten years of age or not until after thirteen years, the average being twelve years. The sixth-year molars, lower central incisors and upper central incisors appear in the order named, but the remaining teeth follow no regular order. When the deciduous tooth has been lost by early decay or extraction the corresponding permanent tooth is apt to appear earlier than normally.

**Abnormalities.**—The cutting edge of functionally normal permanent incisors may show numerous serrations, saw-tooth deformity, with no loss of enamel. These digitations are sometimes mistaken for the deformity described by Hutchinson as a stigma of congenital syphilis.

Periods of undernutrition in the first five years of life are responsible for defects and hypoplasia (defective calcification) in the first group of permanent teeth (incisors). In certain children the teeth show mottling of the enamel with discoloration of the affected spots. This occurs following scarlet fever, measles and any other fever of similar duration and severity. Constitutional diseases and undernutrition are the causes of chalki-

ness and the serrations of the cutting edges, horizontal bands, black stains and pitting. The occurrence of infection affects the formation of all teeth. It is possible that fever stimulates dentition so that eruption is hastened during such an illness.

Occasionally one sees a crumbling of the top of the crown, with a loss of

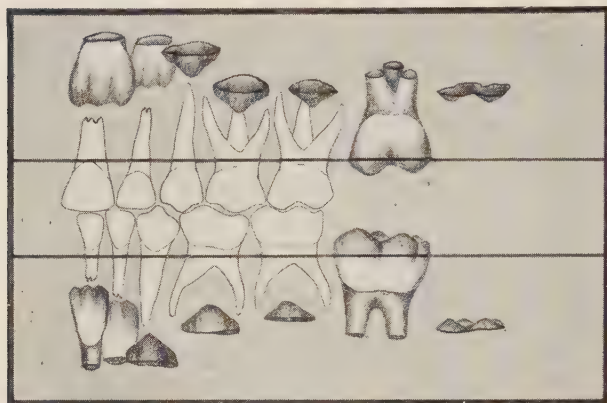


FIG. 17.—THE TEETH AT SIX YEARS

the enamel, the lower half of the crown being of the normal size. This gives the appearance known as the collar-and-sleeve deformity, which affects the incisors and sixth-year molars.

**Caries.**—It has long been known that the primitive races living on coarse foods seldom have caries, but the teeth suffer when the

food of civilization is used. This is well illustrated in the Negro race in our own country. Gibbs calls attention to the Maori race of New Zealand whose children eating a native diet have only 1 per cent of caries, while 95 per cent of those who have been subject to European civilization have bad teeth.

Caries has been progressively more common in Scotland in the last fifty years. Caries and pyorrhea are primarily due to the diet and can be prevented. Prophylaxis is more effective than remedial treatment. Breast-fed infants as a rule have better dental arches and the teeth are more regular and better calcified. Markedly defective diet is responsible for decay.

The diet at weaning should be less of the prevalent soft, sweet, milky foods and more of meat, ripe fruit and fibrous vegetables, which call forth more vigorous chewing. Mastication is the best preventive of dental caries. The practice of giving the older infant the bone of chicken or beef, crusts of bread, or hard crackers does much

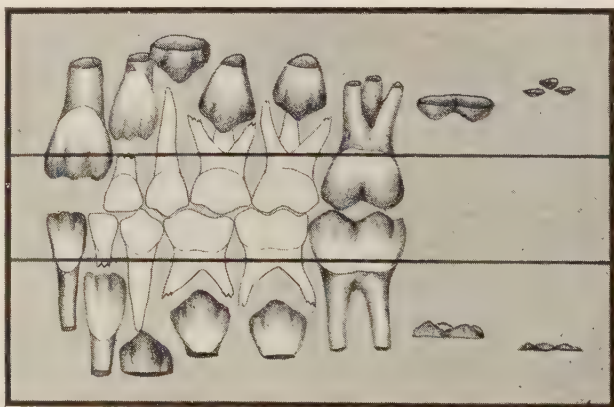


FIG. 18.—THE TEETH AT EIGHT YEARS

to develop the jaws. Freshly popped corn is a valuable food for children beyond the age of three years.

Protein and fat are usually insufficiently fed in the modern diet of older infants and young children. Bread should be coarse, dry and hard. Sweets should not be concentrated but are best taken in ripe, raw fruit, especially oranges and apples. These increase the appetite. Gibbs urges that dental caries may be prevented by having the mouth free of sweets at the end of the meal, using coarse rough foods which assist in cleaning the teeth. Mothers frequently ask if they may give a bar of chocolate; the child's appetite and digestion are better without it, but the mouth should be carefully cleaned after it is used.

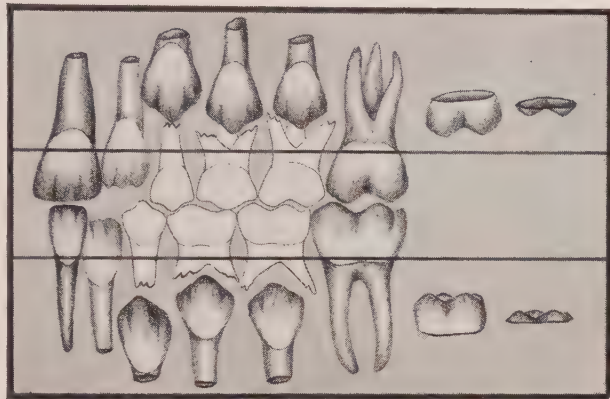


FIG. 19.—THE TEETH AT TEN YEARS

In later childhood proper diet is still necessary for healthy teeth.

Outdoor life and sunshine exert a large influence upon the calcium deposits, a factor which we are apt to overlook.

Carious teeth are frequently the cause of glandular enlargement. Eradication of dental infections will reduce the incidence of glandular hyper-

plasia, especially in school children. There are sometimes long-continued toxemia and acute systemic infection. The writer recalls a child who had been acutely ill for weeks with fever and enlarged spleen, the course much resembling typhoid fever. A discharging sinus at the gum revealed the source of the infection, the extrac-

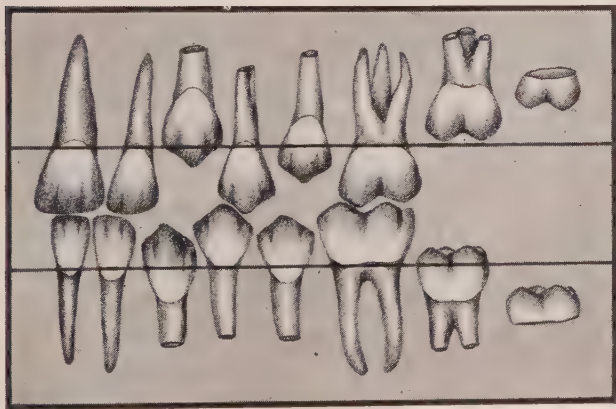


FIG. 20.—THE TEETH AT TWELVE YEARS

tion of the tooth resulted in immediate improvement and cessation of the fever. Toothache and sensitive teeth interfere with chewing and eating, especially of solid food.



Live teeth should be kept filled. Caries should be treated early by the dentist. Teeth which have turned dark are dead and should be pulled.

**Trauma.**—Children's teeth are occasionally traumatized by falls and blows. Such injury may cause devitalization or the tooth may be broken

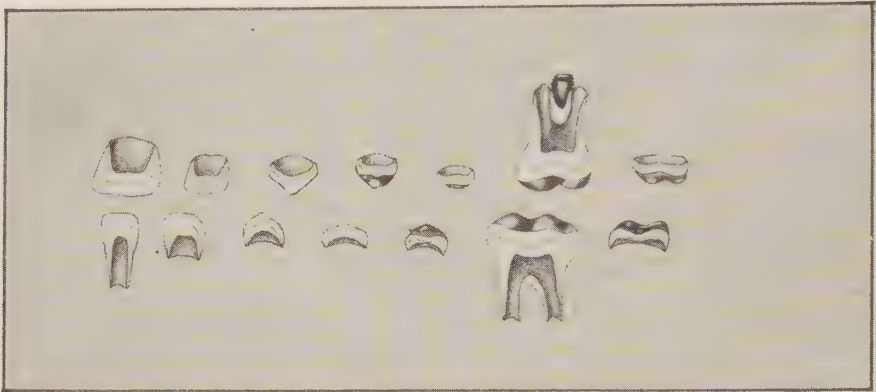


FIG. 21.—PULP DEVELOPMENT AT SIX YEARS

off. If the pulp is uninjured the tooth should be left alone until the roots are mature, when the deformity may be built up by the dentist.

**Malocclusion.**—Malocclusion is mainly dependent upon early caries with the premature loss of the deciduous teeth. It is a serious deformity from a cosmetic as well as a digestive standpoint. Some orthodontists begin the



FIG. 22.—PULP DEVELOPMENT AT TWELVE YEARS

straightening before the permanent teeth have appeared. Early attempts are made to prevent decay, to provide free nasal ventilation by removal of obstructive tonsils and adenoids, and to restrain the child from thumb sucking. Simple appliances may be used for correcting malocclusion in the primary teeth. But it should be remembered that a large percentage of malocclusion is preventable by proper care and retention of deciduous teeth.

This deformity in some degree affects fully half of all children, but only comparatively few ever receive any corrective treatment.

The treatment of malocclusion of the permanent teeth should be started before irremediable deformity of the jaw has developed. The physician should send the child to the orthodontist as soon as irregularities are found. It is not advisable to wait until the child is twelve or fourteen years of age.

**Syphilis of the Permanent Teeth.**—The Hutchinson deformity affects the two upper central permanent incisors. It consists of a central notching or cupping of the cutting edge with a loss of the enamel in this region. Much difference of opinion exists as to whether this dental lesion is specific for syphilis, many observers believing that acute illness or nutritional disturbance at the time of development is responsible. The nutritional disturbance may be due to congenital syphilis in its active stage. When the notching is in the upper lateral incisors the cause while congenital may be due to some other condition. Hutchinson's teeth owe their changes to hypoplasia and to syphilis of the premaxillary bone. The gums also show syphilitic changes by their concave shape which results from this disease in the bony sockets of the teeth, a recent clinical observation which Buschke and Langer regard as diagnostic.

Anterior-posterior thickening of the upper central incisors is pathognomonic of syphilis.

I have occasionally seen a single notching of one incisor in children who were not syphilitic. There are other malformations of the front teeth which have long been considered syphilitic stigmata: for instance the undersized, malformed peg-shaped teeth (chisel-shaped) which are narrower at the cutting edge than at the gum line, instead of being normally wide at the cutting edge. This congenital deformity is not confined to syphilis.

It is possible that certain defects in the enamel of the permanent lower incisors may be the result of syphilis. The nutrition of the enamel or dentine may be disturbed. The sixth-year molars may show early crumbling, irregularity and their surface much depressed below the line of the other teeth.

In cases of complete absence of some of the permanent teeth there has been a failure of development of the dental buds. In such instances the primary teeth may remain unduly in place. The absence of certain permanent teeth and the persistence of corresponding deciduous ones furnish an important sign of congenital syphilis.

## DENTAL HYGIENE

The physician should include a careful inspection of the gums, teeth and glands in his complete examination of children. He will thus become adept in recognizing oral disease and its results, and his opinion will be given due consideration. The attitude of the dentist towards decayed teeth is

often unduly influenced by his very worthy desire to allow adequate growth of the jaws before teeth are removed. Prophylaxis of caries and infection is of much greater importance in permitting normal growth of the mouth, and should be the aim of the dentist and physician rather than the retention of teeth which are dead or have become hopelessly diseased.

Close coöperation between physicians and dentists will result in the better care of the teeth. Too infrequently do the members of these two professions consult about the teeth of the child. For years the writer has advised the parents to take their children to the dentist at three to six months intervals. I have also sought the services of those dentists who are willing to give as much time to the child as is needed. There is a tendency for us to criticize the dentist for his point of view and for what seems to us a lack of interest in the deciduous teeth. On our part, physicians are apt to leave the question entirely alone, as being out of our province. The physician sees the child earlier and more frequently than does the dentist, and his opportunity for observation is greater.

**Prenatal Care.**—As the first set of teeth begins to develop at the fourth month of fetal life, attention should be paid to the mother's nutrition throughout pregnancy. The diet should contain plenty of nourishing food which contains calcium, phosphorus and iron. The blood should be examined, and if anemia or syphilis be present, appropriate treatment should be kept up. Typical Hutchinson's teeth are diagnostic, but present in only a small percentage of syphilitic children. Syphilis can be prevented in the fetus by maternal treatment. This is now common in Scotland and is practiced to some extent in the prenatal clinics in this country. By the universal treatment of the syphilitic mother while she is carrying the child, congenital syphilis will become a rarity. There is no greater misfortune to a child than to be marked with the obvious stigma of Hutchinson's teeth.

**The Preschool Child.**—There is a gradually increasing tendency on the part of thoughtful parents to give their young children adequate supervision by a dentist, and to establish regular habits of mouth cleanliness. Little attention has as yet been paid to the kind of diet which has the best effect upon the teeth. The diet is of great importance. At the Forsyth Infirmary in Boston, one of the most advanced dental research institutions, the following articles of food are recommended for increasing and maintaining the nutrition of the teeth:

Cracked wheat	Irish oatmeal	Molasses
Cracked corn	Wholewheat bread	Honey
Hominy grits	Oatmeal bran	Maple sugar
Brown rice	Bran (rye)	No white sugar

Thumb sucking and pacifiers when persistently used cause protrusion of the upper teeth. This disgusting practice is slowly becoming obsolete. The first appearance of even small cavities should be the occasion of careful

dentistry, as the filling of these areas will tend to prevent suppurating roots. The early loss of the deciduous teeth favors the early eruption of the permanent ones before the jaw is sufficiently large. As badly decayed teeth must be extracted, we should emphasize the *prevention* of caries.

Visits to the dentist at this age and at stated periods teach the child the necessity for taking care of the teeth and give the family the opportunity for safeguarding the permanent teeth from the time of eruption. In most large communities dentists may be found who will undertake the prophylactic care of the deciduous teeth. Every effort should be made to prevent nutritional disturbances which might cause lowered resistance, infection and deficient calcification of the teeth.

**The Permanent Teeth.**—The child should be trained in the habit of cleaning the teeth after each meal. A glass of warm water as a mouth wash and a firm tooth brush to clean the gum margins and the dental interspaces will be of value. I am not very optimistic about the probability of children using the tooth brush in a thorough manner. As decay usually results from decomposing food between the teeth and under the gums, and as such foreign material is usually ineffectually removed with the brush, other methods of cleansing may be tried. The mother may use a rubber band or piece of dental floss drawn between the teeth. Prophylactic care is the most important. Cavities should be filled carefully and permanently at the first appearance while the caries is yet slight.

The diet is important at this age. The secretion of the saliva should be encouraged by the use of acid foods, fruits, vegetables, salads, dry and coarse breads.

As the roots of the sixth-year molars are not complete until the child is nine to fourteen years of age, the death of the pulp before the roots are completed means their complete loss, as it is then impossible to fill the roots.

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## CHAPTER VII

### THE ESOPHAGUS

**Congenital Malformation.**—Entire absence of the esophagus is rare, as is also such a marked deformity as bifurcation.

*Congenital atresia* or failure of development of a portion is being more frequently reported, and is probably not so unusual as the occasional mention

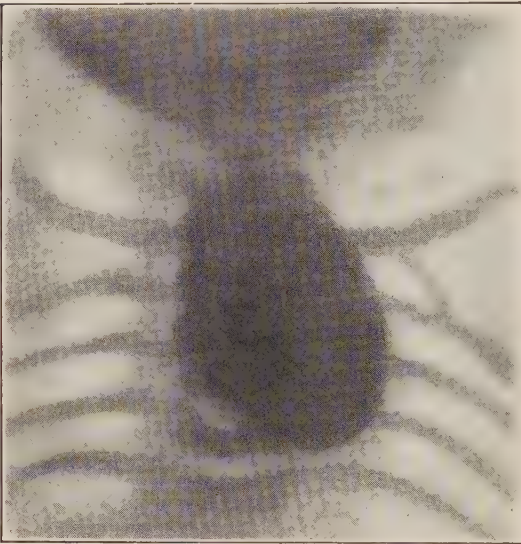


FIG. 23.—CONGENITAL ABSENCE OF MIDDLE PORTION OF THE ESOPHAGUS

The opaque area in the center of this picture represents the upper portion of the esophagus filled with barium and ending in a culdesac at the level of the fourth dorsal vertebra. (F. C. Neff, *Am. J. Dis. Child.*, 1921, 22: 57. Reproduction by permission of the publishers.)

in the earlier literature would indicate. About 217 cases have thus far been collected. Brennemann, in 1918, reported seven cases of his own. I have seen three cases. Such a malformation is incompatible with life and adds considerably to the mortality of the newly born period. The infant immediately vomits its water or food, but the fact that none reaches the stomach may be unknown to the attendants until the fact of complete obstruction is recognized.

Various types of esophageal malformation are described by Ballantyne, the most common form comprising three-fourths of the cases. This is an interruption of the continuity of the

esophagus, with the lower or gastric portion communicating with the trachea or bronchus. There is then an absence of the middle portion of the esophagus, the upper third ending in a blind pouch or culdesac; the lower or gastric portion communicates with the stomach below but the upper end becomes smaller until only a narrow but patent cord reaches and communicates with the respiratory tract. This is called the inosculating type.

In other less common forms the gastric portion of the esophagus is projected upward into a fibrous cord which approaches the bronchus or may

unite the two segments of the esophagus. Complete absence of the middle portion with no tracheal or bronchial communication occurred in a case reported by the writer. This variety corresponds to No. VII (b) in the classification of Cautley. The upper portion ended in the usual culdesac and the walls were thick. The lower portion extended upward from the

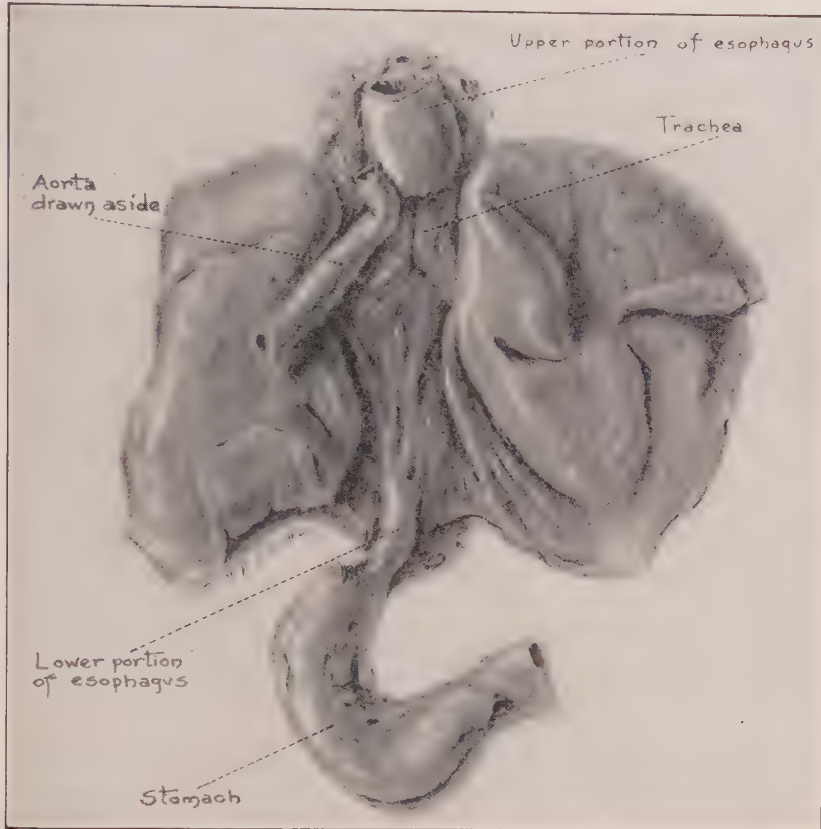


FIG. 24.—ATRESIA OF THE ESOPHAGUS

The upper portion of the esophagus which ends blindly measures 2 centimeters in length by 1.2 centimeters in width and the lumen 0.7 centimeter. The lower portion extends from the cardia upward in branched prolongations, the middle portion absent. There is no communication between the two, nor with the bronchus or trachea. The lower part of the esophagus is patent for less than 2 centimeters.

cardia, was patulous for only 2 centimeters and became lost in various fibrous prolongations in the mediastinal tissue.

*Etiology.*—No satisfactory explanation for this failure of development has as yet been offered. The esophagus and respiratory tree develop from a common origin in the foregut during embryonal life. The failure of these to completely separate is responsible for the anomaly.

The association with other malformations of the intestinal tract is reported, the most commonly atresia of the anus.

*Symptoms.*—The manifestations consist of prompt vomiting of all intake, rapid wasting and starvation. Attempts at feeding may cause

violent attacks of coughing, due to entrance of food into the larynx. The temperature may be below normal in the beginning, but fever accompanies complications. Death occurs in the second week from starvation and from the pneumonia developing after aspiration of vomited material.

A case seen by the writer illustrates the fact that esophageal atresia is recognized with more difficulty in the premature than in the vigorous infant at birth. This child weighed 4 pounds 11 ounces on the first day and was too weak to attempt nursing. It was fed with a spoon a small portion of breast milk which was immediately vomited. This intolerance for food was ascribed to prematurity of the stomach and to weakness and it was not suspected at first that all of the intake was being lost. At the end of one week, the weight was 25 per cent below that of birth. Attempt to feed with esophageal catheter disclosed an obstruction at a distance of 4 inches from the gums and this was confirmed by a barium radiogram. The body temperature was always above normal. This congenital weakling lived without any food for eight days. Autopsy revealed an upper blind pouch united by a fibrous cord into a patent lower portion of the esophagus. There was no communication between the esophagus and the respiratory tract. The lungs contained areas of pneumonic and hemorrhagic infiltration.

*Treatment.*—No operation has as yet been successful, though attempts have been made to unite the upper and lower portions of the esophagus. Gastrotomies have likewise been unavailing. In one of my cases, a gastric fistula was made, through which the infant was fed for a few days. Death occurred from hemorrhage and inanition.

**Congenital Narrowing.**—In older children, especially from two to three years of age, repeated vomiting since early infancy may be due to partial constriction or narrowing of the esophagus. There is usually a dilated portion just above the stenosis. Fluids will pass usually, but solid food and mucus accumulate in the dilated part. Because of the vomiting and lack of retention, the nutrition suffers. Morse reported three such cases in which the esophagus showed fusiform dilatation above the congenital narrowing. Strauss and Hess recently described a type of stenosis, congenital in origin, occurring just above the diaphragm with a proximal dilatation and with other associated malformations such as pyloric stenosis and a cecum which was not rotated but ran in a straight line with the colon from the liver to the rectum.

The treatment in the type described by Morse is successful by the production of gradual dilatation with bougies. Strauss and Hess warn against the attempt to dilate the type of stricture which they have described. They recommend operation by exposing the cardiac portion of the stomach, when by freeing the bands which unite it to the diaphragm, the esophagus may be brought down sufficiently to incise its outer coats and constricting muscles.

**Diseases of the Esophagus.**—Abnormal conditions in the esophagus during childhood are not infrequent, as is shown from the recent advances in their recognition and relief. The esophagus is especially exposed and

susceptible to injury and infection, a fact that is better appreciated since the use of roentgenoscopy and electrically illuminated instruments have revolutionized the diagnosis and treatment.

The two most common serious disturbances in the esophagus during childhood are the lodgment of a foreign body and the injury from caustic alkali burns, both of which are the result of accident. The incidence of the former will probably be little reduced, but an effort is being made to prevent the occurrence of the latter. Acute and chronic esophagitis is rare from any other causes than these.

As a result of specialization in this field which is highly technical, accidents to the esophagus are being more frequently referred to the well-equipped and experienced clinician in this line. The wonderful work of Chevalier Jackson and his coworkers shows the value of a special diagnostic and therapeutic clinic to which come many cases from a distance. It would be well, if in large centers of population, all work of this kind were referred to one group, for the number of cases is insufficient to give the necessary experience to all the surgeons and throat specialists who now undertake it.

*Anatomy and Physiology.*—The shape, structure and function of the esophagus have much to do with the occurrence of lesions. The walls are delicate and vulnerable, though the mucous membrane in infancy is proportionately heavier than the muscularis. The presence of many kinds of bacteria makes secondary infection easy. Jackson regards the esophagus as particularly intolerant of surgical exploration.

The upper and lower boundaries are placed higher with reference to the vertebræ than in adults. In the infant these limits are the fourth cervical and the ninth dorsal vertebræ.

The average diameter and the length from the incisor margin to the cardiac opening are given in the following figures adapted from the table of Scammon:

Age	Diameter, millimeters	Distance from Incisor to Cardiac End of Stomach, centimeters
Under 2 months .....	5 to 8	18
3 to 8 months .....	9	20
9 to 24 months .....	10	23
6 to 12 years .....	12	26 to 30

The diameter of the lumen is constricted at four levels so that a rigid metal tube must be small enough to pass these constrictions, varying from 7 millimeters in infants to 10 millimeters in the adult.

The walls of the esophagus move during respiration and from pulsation of the aorta. The chief movements are due to contraction connected with swallowing or vomiting. The passage of food is furthered by the peristaltic



wave in the esophageal wall. The normal prevention of gastric regurgitation is due to the closure of the muscles of the diaphragm, and to kinking of the abdominal portion of the esophagus. There is no valve or sphincter in the lower end of the esophagus.

**Foreign Bodies in the Esophagus.**—*Incidence.*—Because of the tendency of infants and young children to carry to the mouth any object which they grasp, accidental swallowing of such not infrequently happens. It is

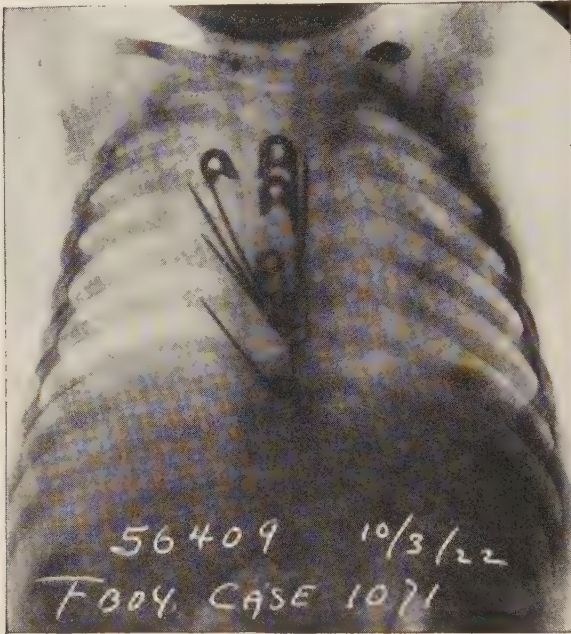


FIG. 25.—FOUR OPEN SAFETY PINS IN THE ESOPHAGUS  
(Reported by Chevalier Jackson, *J. Am. M. Ass.*, 1923, 81: 1099, and loaned by courtesy of the author and journal.)

not to be questioned that the safety pin is the most common and the most dangerous. The remarkable fact is that objects whose size greatly exceeds the diameter of the tube may successfully pass. They are not so easily regurgitated.

Small objects which are sharp or rough show more tendency to lodge than do large smooth ones. Dickson reported a remarkable instance of a child who swallowed a thin padlock, 5 centimeters in its greatest diameter, which was passed within two days. The weight, smoothness of the object, and an un-

usually large alimentary canal must have been factors in making such a feat possible.

As many as four open safety pins were found in the esophagus of a nine-months-old child, with interlocking so that the pins could not be removed except by pushing two of them into the stomach and these passed easily through the stomach and bowel; the upper two were removed by aid of the esophagoscope through the mouth. Although the pins had been in the esophagus for two months, the child made a normal recovery.

Occasionally an article of insignificant size but with rough or sharp exterior may lodge on the esophageal wall, a common illustration of which is the sand bur and fish bone. In one case a small crust of bread remained lodged for one week.

**Lodgment of Safety Pin.**—Closed pins easily traverse the esophagus. Open pins if small usually pass, even when point downward. I have known

of the appearance of such in the stool within three days and without any untoward symptoms. Open safety pins, however, are apt to become fast in the esophagus when point is downward because of the outward pressure of the spring. The curled end of the spring is more liable to be foremost in the course of swallowing, the open end becomes engaged in the wall of the pharynx and the curled end may swing around and start downward. An open pin with the point upward is difficult to withdraw unless the pin be closed *in situ*. It is sometimes necessary to push it into the stomach, where it may be grasped by the esophageal forceps, turned and withdrawn, or allowed to pass by the peristaltic action of the stomach and bowel.

*Symptoms.*—The lodgment of the foreign body does not always produce symptoms. The swallowing may be accompanied by some difficulty, discomfort, pain or hemorrhage. At the time of ingestion, there is usually a choking or retching sensation, and in older children the feeling of incomplete swallowing. The sensation persists for awhile after the swallowing or removal of the object, if this has been difficult. Injury of the esophageal wall may result in bleeding and in elevation of temperature.

The unsuspected presence of a safety pin in the esophagus caused vomiting of blood for a period of six months in a two-year-old child reported by Jackson. The pin had produced granulations and swelling of the esophageal lining, obstruction, and a general anemia. Removal of the pin through the mouth resulted in cure.

Dysphagia is the symptom most apt to be present. Any retained foreign body interferes with coarse food and, by the resulting spasm, even with the swallowing of liquids. The usual site of obstruction is in the region of the anatomical constrictions of the esophagus or occasionally at the seat of a diverticulum or a congenital stenosis.

The presence of pain depends upon the amount of injury to the wall. Marked distention, inflammation or piercing of the wall may cause pain. This may be substernal or referred to the back.

Vomiting, regurgitation of food or of accumulated saliva and mucus occur in most cases, especially if the obstruction is severe. Blood may be present. The child prefers to lie in the prone position.

Severe cases may have edema, inflammation and ulceration of the esophagus. Unsuccessful attempts at removal cause marked increase of symptoms. Perforation of the wall is the most probable, and this may cause hemorrhage and suppuration. The most serious injury is rupture into the pleural cavity, or through the posterior wall of the esophagus with resulting infection of the mediastinum and the development of general sepsis. Untreated cases all die from subsequent abscess, fistula and starvation.

*Complications.*—Regurgitation of fluids from the obstructed esophagus may be aspirated through the glottis and cause respiratory infection. Interference with respiration may be caused by direct pressure, trauma or perforation from the foreign body or by examination. One unusual compli-

cation which I saw in the practice of Sam Roberts concerned a safety pin which had slowly worked its way into the tissues between the esophagus and trachea, remained quietly buried there, and was removed by incision and dissection from the side of the neck.

Perforation of the wall of the esophagus from the presence of a foreign body such as a bone or pin may result in abscess of the surrounding tissues. Minnigerode has noted the early diagnostic value of a roentgenogram in detecting a leak in the wall at the site of the foreign body. The air escaping from the wound or the presence of gas causes a light strip at the perforation. The early detection of this serious complication permits of successful treatment.

Incredibly long and sharp objects may traverse the esophagus and pass through the stomach. A hat pin, more than two inches in length, reported by Skirving, was swallowed by a three-year-old girl, and produced no pain. It finally perforated the duodenum from which it was removed by laparotomy.

*Examination.*—The beginning of the examination should consist of a clear inspection of the mouth, tonsillar fossæ, and pharynx. A laryngeal mirror should be used for inspecting the nasopharynx and esophagus. If the object is seen it should be firmly grasped with a pair of forceps and gently withdrawn. There is often the possibility of the lodgment in these regions. In the case of an open safety pin, special apparatus may be needed. The physician is cautioned against blindly probing with the finger or bougie, or attempting removal with the fingers, as the object may be pushed further down or into the soft structures. The child should not be inverted with the hope of removing the object as it is unsuccessful and not free of danger.

When the foreign body is not visible, the next step should be the use of the fluoroscope and radiogram. It is not easy to determine from an anterior-posterior radiogram whether the object is in the esophagus or larynx, but a lateral or quarter view in addition is conclusive. Pins, coins, and buttons that give a transverse shadow are usually in the esophagus. The entrance to the larynx is slit-like and permits only the narrow edge or end to enter, and it tends to remain in the same relative position. The esophagus is more elastic. When the obstruction is due to an object which is not opaque, Jackson recommends the swallowing of a barium mixture or capsule which will lodge at the site of obstruction.

With the localization of the foreign body, the physician who is not equipped and experienced will do well to seek the advice and technical assistance of a laryngologist who has become skillful in this line of work. Esophageal obstruction should be first examined under direct illumination through an esophagoscope, and the method of removal decided upon.

*Diagnosis.*—The diagnosis is made by the history, difficulty or pain in swallowing, vomiting, and by absence of physical signs in the air passages. The subjective symptoms and the character of interference with the function



of the esophagus do not always give an indication of the location of the foreign body. When the physical signs and the history are negative, foreign bodies are overlooked until the routine examination of the child who is not eating well, or is losing weight, suggests the use of the roentgen ray for diagnosis.

Opaque objects are clearly seen by the roentgen ray whether in the esophagus or air passages. Barium cannot be used in the respiratory tract. The objects which are most difficult to visualize in the bronchus, namely, peanut kernels, beans, and other vegetable substances, are not the cause of esophageal obstruction.

The diagnosis as between respiratory and esophageal foreign bodies is usually not difficult. The swallowing of a foreign body calls for an immediate decision as to whether the larynx or the esophagus has been entered. Choking, gagging, coughing and wheezing attacks mark the entrance into the larynx, which may be followed by hoarseness and inability to speak or cry, wheezy or croupy, painful or difficult breathing and temporary cyanosis. The occurrence of complete obstruction is rapidly fatal. The direct examination of the larynx should be made as soon as possible before the object has time to pass lower. In the tracheal obstruction a slapping sound may be heard while listening at the open mouth while the patient is coughing. In the same way the typical "asthmatoïd wheeze" (Jackson) may be heard during breathing. The physical signs in bronchial obstruction may not be constant, but will usually show a lessened expansion on the affected side, diminished breath sounds and vocal fremitus. In complete obstruction, râles are more numerous on the well side, but in incomplete obstruction they are on the affected side. The percussion note is tympanitic while emphysema is present, but becomes dull after the collapse of the lung.

The physician should exercise care in giving an opinion as to the absence of a foreign body in the esophagus until all necessary diagnostic efforts have been made.

*Treatment.*—Great advance has been made in the saving of life. When the diagnosis is known, the child should be at once referred to an expert in esophagoscopy. No temporizing with coarse foods, emetics or probing is warranted. The child should have a careful examination to exclude the presence of foreign body elsewhere. The stools should be saved for careful examination by the physician. Fluoroscopy is to be used, and pictures taken at various angles may be useful.

No drugs are indicated, but a hypodermic injection of codein or morphin may be required for severe pain.

Trauma is increased by attempts to push downward the foreign body, and rupture of the esophagus may occur. A safety pin can often be closed, and withdrawn in safety. The wisest course is the use of direct illumination, and the withdrawal upward of the object when this is possible.



The mortality in uncomplicated cases by skillful early removal is 2 per cent, but much higher by the older blind methods. The opening of the esophagus is dangerous and seldom necessary, as the foreign body can be removed by direct methods. Perforation and suppuration into the neck or chest require early incision and drainage.

**Spasm of the Esophagus.**—The types of esophageal spasm vary with the cause. When it is primary it is of neurotic origin, and may be periodic or constant, affecting part or all of the esophagus. Emotional spasm causes vomiting in a child who is fed against his will. There has been described in Brazil a type of spasm which is probably epidemic, and occurs in the portion of the esophagus below the diaphragm. This dysphagia is found in infancy as well as at other ages, is recurrent and may cause death through inanition.

The normal movements are restricted by a spasm of the wall which may be produced by any irritation such as the presence of an unusually large bolus of food, the lodgment of a foreign body, or the development of edema, inflammation, or ulceration of the mucosa.

So-called *cardiospasm* is, according to Jackson, impossible at the cardiac orifice. A functional closing at the level of the diaphragm does occur at times from extrinsic cause, occasioned by the "pinch cock" action of the muscles of the crura at the diaphragmatic opening. Above the constriction the esophagus becomes dilated. The symptoms are belching, frequent vomiting, or, when a large accumulation occurs in the dilated portion a sensation of pressure behind the sternum, and impairment of the child's nutrition. The diagnosis is made by the failure of barium to pass, but a rigid tube will pass the obstruction without difficulty.

The treatment is daily lavage of the dilated portion, and feeding through a stomach tube. The functional stenosis is cured, sooner or later, by the dilatation resulting from passing the tube.

**Stricture of Esophagus.**—The accidental swallowing of acid or caustic substances, especially concentrated lye, is not an infrequent occurrence. Through carelessness the young child gets hold of an open can or solution of lye or other strong preparation of sodium hydroxid which is commonly kept in the household. Ingestion causes necrosis of the esophageal wall, death, or in cases which survive, a partial or complete cicatricial stenosis.

Other causes occasionally lead to scar formation in the esophagus. Ulceration of the esophagus can come from pyogenic and other infections, and these lead to more or less stenosis. The irritation and continued presence of foreign bodies, spasm of the wall with retained food and micro-organisms, trauma from sharp objects, and instrumentation, if not gentle, will produce partial obstruction.

**Pathology.**—Stricture is most common in the portion that is crossed by the left bronchus. More than one stricture is often present. When the scar formation on the sides is irregular, the walls are distorted and saccu-

lations or pockets are formed. The axis of the lumen may therefore be tortuous, unless the cicatrix involves the whole circumference. The passage of a catheter or sound is difficult, in some cases impossible unless the opening is visualized. Extensive scar formation interferes with the movements of the esophagus. Dilatation occurs above the stricture.

The strength and quantity of the poison influence directly the extent of the tissue destruction, which varies from slight inflammation and ulceration to necrosis of part or all of the thickness of the wall, and perforation. Marked inflammatory reaction surrounds the burned areas. Following perforation, infection by some of the many microorganisms present in the esophagus leads to peri-esophageal inflammation and suppuration which spreads downward to the thorax, causing mediastinal and pleural abscess.

The difficulty in taking food is due to pain, spasm of the walls, inflammatory infiltration, scar formation in the submucosa with resulting stricture.

*Symptoms and Course.*—The early symptoms are those of the corrosive action of the poison upon the superficial strictures of the mouth, pharynx and esophagus. Pain, spasm of the throat muscles, and difficult swallowing occur promptly. There is retching and vomiting of mucus and blood. The mucosa is markedly reddened and soon denuded. Shreds of mucous membrane are vomited in severe burns. Extensive tissue necrosis causes shock from which the child may die promptly or within a few days, during which period the child is stuporous and develops fever. Gaizler ascribes the presence of acetone in the urine to the destruction of protein in the necrotic area. Necrosis of the blood-vessels may cause severe hemorrhage, and the surrounding structures of the neck and mediastinum involved in the sloughing process. Intoxication is marked. Early death occurs in many cases, and is foretold by the extreme degree of burn in the mouth and throat.

In less severe cases the child recovers from the early collapse, but there is marked inflammatory reaction in the mouth and esophagus, upon the amount of which the severity of the symptoms depends. The course is distressing to an extent found in few other conditions, and even where it does not end in death it means a long period of invalidism, with dysphagia, starvation and obstruction steadily progressing in the untreated case. Development of perforation and suppuration is a serious sequence, followed by phlegmonous lesions and general sepsis. The cases that live, nearly always have more or less cicatricial formation in and around the esophagus which interferes with the function of the food passage.

When stricture develops it may soon prevent the passage of coarse food, and in severe cases not even fluids may go through. The accumulation of food masses will obstruct the lumen at the site of the stenosis. Regurgitation and overflow of the contents of the esophagus occurs at times. Distention of the esophagus causes discomfort. There is apt to be swelling of the mucosa and an associated spasm which causes intermittent obstruction. The interference with the swallowing of sufficient food causes the chief



FIG. 26.—STRICTURE OF ESOPHAGUS FOLLOWING LYE BURN

Although the stricture was almost complete sufficient barium passed so that the stomach is well coated with barium. Not enough of the opaque substance remained in the strictured portion to cast a shadow.

embarrassment to survival, for the weight becomes progressively lower, and inanition is imminent.

*Diagnosis.*—When the stenosis is complete all food is vomited, but even rather thick food may pass at times through a fairly tight stricture. The radiogram is a necessity in locating or in determining the degree of patency. Radiograms are not always satisfactory, because of their failure to give a satisfactory outline of the lumen. Pollerman has advised the use of a bismuth mixture which is better retained by the esophagus. He uses bismuth subcarbonate with 8 parts of oil of bitter almond, 8 of water, 4 of gum arabic, well shaken at time of administration.

*Prognosis.*—Van Hacker gave the prognosis of alkali burns of the esophagus as fatal in 25 per cent, mild stricture in 25 per cent, and serious stricture in 50 per cent. Guisez (Paris) reported recovery in thirty-five of forty cases treated by various methods. Three deaths were early, and two died later with recurrent stricture.

*Prophylaxis.*—The family physician will render a definite service to his clientele if he warns the family of the danger of keeping poisons in the home. The opened can of concentrated lye, or the utensils which have contained solutions of lye or washing powders are too often carelessly left accessible to the child.

The prevention of this type of accident has received the attention of the American Medical Association which has urged legislation towards safeguarding the public. Proper labeling, the use of containers which are difficult to open by children, removal of the cans from accessibility, the destruction of the remainder of the contents, the thorough rinsing of all household vessels, but more important still, the propaganda against the purchase of all dangerous substances, are methods which should be followed. No home with children should tolerate the use or presence of as dangerous a substance as lye.

*Immediate Treatment.*—Antidotes.—It is usually impossible to administer neutralizing remedies sufficiently early to be of much service. In case of lye poisoning, lemon juice should be at once given in large quantities. The difficulty of swallowing may prevent any antidote from reaching the esophagus. Chalk water and milk of magnesia are indicated in acid poisoning, whisky and grain alcohol solutions for phenol and lysol poisoning.

The chief indication is the relief of pain by hypodermic injection of codein or morphin. Water or crushed ice is needed for the thirst. For shock, external heat and hypodermics of epinephrin solution are to be used.

The prevention of severe stricture after swallowing lye is regarded as possible and advisable. In 56 cases treated by Salzer and 147 by von Bokay lye erosions were treated early, on the second day in mild burns and on the sixth day after severe ones. The method used is the gentle introduction of a No. 30 to 32 rubber bougie filled with shot to carry it by its weight into the stomach. The bougie is placed in hot water until softened. For the



first three or four weeks the bougie is introduced daily, for a few minutes, then less frequently but for a longer period, and repeated at weekly intervals until cure is established. The results are good, and usually no functional stricture is formed.

Probably most cases are now treated by the threaded guide, and gradual dilatation produced. When this is easily possible, no objection is found to

such a method. Children are not such good subjects for voluntary swallowing and retention of thread.

*Feeding.*—In total obstruction of the esophagus from cicatrization, no saliva reaches the stomach. In order to feed the child a gastric fistula must be made. Such children do not thrive well when fed through the gastrotomy opening, due, as found by Jackson and his coworkers, to the absence of salivary digestion (starch-digesting enzyme in the saliva).

By a method devised by these workers, the child spits into a funnel connected with the gastrotomy tube. This enters the stomach and becomes mixed with the gastric meal. As soon as the esophagus can be made patulous, saliva will enter the stomach in the natural way.



FIG. 27.—INSALIVATION OF FOOD THROUGH GASTROTOMY WOUND

(Courtesy of Chevalier Jackson and *Arch. Pediat.*)

*Early Dietary.*—The nutrition suffers from the beginning because of the impossibility of taking the complete dietary necessary for the child. The child should be given fluids exclusively. These should be milk and strained liquids, preferably the juices from fruit and beef and vegetable soups. If thin gruel will pass the stenosis, it should be given. In case of doubt as to whether food is passing, the fluoroscope will be of help in determining the patency of the lumen. Observation should be made at regular intervals.

A method which makes possible both the regular maintenance of feeding from the onset and the prevention of marked obstruction has been used by Roux since 1913. Immediately following the accident a soft rubber hollow catheter (Nélaton) 8 to 11 millimeters in diameter, with a length greater

than the ordinary catheter, is lubricated and passed into the stomach. If this is impossible, due to the esophageal spasm, an anesthetic is given. The stomach is washed out, and the tube left continuously in place for three weeks. Discomfort is said to disappear after a few days. Feeding through the tube is begun on the first day. Roux believes absorption and intoxication from the poison is decreased by the early lavage of the stomach, and that the tendency to perforation and pyloric stenosis may be lessened. This method is worthy of trial in cases that are inaccessible to other forms of early treatment.

*Instrumental Treatment.*—Because of the better results secured by the recent methods of treatment by aid of the esophagoscope it is recommended that the child be placed in the hospital under the care of the special surgeon whose experience and competency is recognized. The average physician sees so few of these cases that he cannot be skilled in this line, nor equipped with the necessary apparatus. We should welcome the opportunity to refer these children to others who are in a position to give them the best opportunity for life and cure.

Gastrotomy is the immediate treatment for severe stenosis that prevents intake of food. As soon as feeding is established through the fistula, and the nutritional needs supplied, local treatment of the stricture may be begun.

The experienced laryngologist will adapt the method of dilatation of the stricture to the needs of the individual case, and will be guided by the radiogram and by direct illumination in his examination and treatment.

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## CHAPTER VIII

### THE STOMACH AND ITS DISTURBANCES

**Anatomy and Physiology.**—The divisions of the stomach are the same in infancy as in adult life. Formerly it was thought that there was no fundus in early infancy, but the roentgen examination and other recent studies show the functioning stomach to possess one. The length of the fundus is relatively shorter in early life and grows faster than the other parts of the stomach.

Much difference of opinion exists as regards the shape, but it is now considered that many factors influence it. The contour varies with the amount of air contained, and with the position of the child, whether it is lying down or being held up. The chief factor however is the presence and weight of the food in the stomach and the consistency of the contents. The contraction of the walls due to peristalsis temporarily changes its shape.

The motility and shape of the infant's stomach are becoming better understood. A new function was described by Epstein in 1920, who noticed a large amount of air in the stomach of infants. For some time it has been known that thick food is less apt to be vomited than liquids. Epstein noticed that when thickened gruel is fed, the stomach adapts its shape to the gastric contents, and thus expels the air. This ability to change its shape and to grasp some semisolid or solid food is called the "peristolic" function. Rogatz has recently published two interesting papers dealing with this newly described function. Thick food cooked with milk tends to become liquefied while cereal or potato prepared with water and boiled to a thick consistency will remain thick in the stomach and thereby be better grasped by the surrounding wall. One therefore must conclude that the shape varies with the nature of the food. When fluids are given the stomach is oval or pear-shaped. When food which remains thick is fed, the stomach assumes a circular shape, is much smaller and contains less air.

The position also varies with age, feeding and body posture. In the fetus the axis is vertical. In early infancy the stomach lies transversely especially when filled, due to the liquid food and probably also to the effect of the usual recumbent posture.

*In Older Childhood.*—After the age of infancy, when the child is up and running around, the position again becomes vertical and the shape gradually assumes that of adult life. The amount of air found in the

stomach and intestine is much greater in infancy than in older children. The change in shape is due to the upright position of the older child, and less distention from air. From the age of six to fifteen years the shape, size and relative position show many variations. In middle childhood the type is usually that of the "sink-drain." In later years it gradually comes to resemble the fishhook or siphon type of adult life. In studying the relative position in the abdomen, Wright found that the lower border of the stomach may be below the level of the iliac crests as frequently as above, but the low position is more common in girls.

The emptying time is not dependent on age but somewhat upon the nature of food, liquids passing more rapidly. Eighty per cent of children have an empty stomach in from one to two hours after eating.

*The Emptying Time in Infancy.*—The stomach empties in from two to three hours, being shorter with fluids, small amounts and with breast milk. It depends upon the gastric tone, and the appetite. When the infant is held against the fluoroscopic screen, a portion of the contents may be observed leaving the pylorus within a few minutes. The emptying of the stomach is favored by gravity when the child is held in the upright position. The time is said to be lengthened by the ingestion of large amounts of sugar and candy at or near the mealtime by decreasing the gastric juice.

*Capacity in Infancy.*—The amount of fluid which can be held by the stomach is difficult to estimate in the live infant, as some food or water has passed through the pylorus by the time the nursing period is ended. Bottle babies take a relatively larger quantity than breast babies, unless the artificial foods have an equal concentration and nutritive value.

The average maximum physiological capacity in breast-fed infants for the first ten days was found by Scammon and Doyle to be as follows:

AVERAGE MAXIMUM AMOUNT TAKEN AT ONE TIME BY THE NEWLY BORN INFANT  
WEIGHING ABOVE 4 POUNDS 7 OUNCES

Day	Grams	Ounces
1 .....	9	0.3
2 .....	22	0.7
3 .....	43	1.4
4 .....	65	2.1
5 .....	78	2.6
6 .....	85	2.8
7 .....	90	3.0
8 .....	94	3.1
9 .....	99	3.3
10 .....	105	3.5

The capacity on the second day is twice that on the first day, nearly five times by the third, seven times by the fourth, but thereafter increases more slowly until at the tenth day it has reached eleven and one-half times the amount on the first day.



In later infancy the average amounts taken are :

Month	Grams	Ounces
1 .....	110	3.6
2 .....	130	4.3
3 .....	150	5.0
4 .....	170	5.6
5 to 12 .....	180 to 270	6 to 9

The formula for estimating the capacity of the stomach for meals from the end of the first month to the end of the sixth month of life has been worked out by Scammon and is as follows :

The capacity in cubic centimeters =  $90 + (5 \text{ times the age in weeks})$ . As an example showing the working of this formula the amount taken by an eight-weeks-old infant is as follows :

$$\text{Capacity} = 90 + (5 \text{ times } 8) = 130 \text{ c.c. } (4\frac{1}{3} \text{ oz.})$$

Generally speaking the *digestive function* of the stomach in the first months of life is relatively small. This is noticed in the frequent inability to handle mature food except of the simplest nature. Gastric digestive upsets are mostly expressed by vomiting. It is easy to overstep the boundaries of tolerance. The organ is immature, not only in size but in its structure. By minute examination there is found to be an infantile development noticeable in the mucous and acid-producing glands, and in the elastic, muscular and nerve fibers of the gastric wall. The mucosa is relatively thicker than the muscular coat. The gastric juice however contains rennin, hydrochloric acid, pepsin, and peptone, but in small amounts. The number of secreting glands in the stomach wall increases rapidly as childhood advances.

It is probable that milk digestion in the stomach is more complete in the newly born infant than in later months. It was found by Griswold and Shohl that the gastric contents in the newly born infant, who had as yet received no food, consisted of free hydrochloric acid. A few years ago incredibly dilute formulas were given to the young babies. For two years I have been feeding newly born infants in the obstetrical nursery, without any digestive disturbance developing, mixtures containing half or two-thirds whole soured milk.

Such intestinal disturbances as enteritis in young children interfere with the gastric digestion. Fermentation is increased when there is less hydrochloric acid produced or when the bacterial content is increased.

In the normal infant the only gas contained in the stomach is air, but in gastric disturbances with fermentation such gases as carbon dioxid, hydrogen and methane are said to be present. In artificially fed infants with butyric acid fermentation the amount of gas may be reduced by the feeding of buttermilk. It is probable that in many of the instances where the mother

concludes that the baby has discomfort from gas, the distention has been produced by excessive air-swallowing. Less air will be swallowed and more expelled by holding the nursing infant in the semi-erect or right lateral position; this also aids food to pass through the pylorus. The emptying of the stomach is hastened by the presence of acid food near the pylorus and is retarded by contents of alkaline reaction. The neutralization of the gastric hydrochloric acid by alkali delays the formation of large curds in the stomach. In certain instances the acid glands may be stimulated to produce increased secretion as a result of the neutralization of the food by artificial means. The gastric motility is very rapid in infancy, as can be readily determined from the emptying time observed under the fluoroscope or by catheterization of the stomach at regular periods.

There is little direct food absorption by the gastric villi. Only a small amount of sugar and digested protein is taken up by the stomach wall. Fat, salts and water pass almost entirely into the intestine. The retention and accumulation of water and all varieties of food in pyloric stenosis, even in the presence of much vomiting, bear out the fact that the stomach has little to do with absorption.

The knowledge of the capacity of the stomach and its emptying time is of practical application in determining the amounts and thereby the frequency of feeding. That infants were formerly successfully fed at two-hour intervals and in recent years at three- or four-hour intervals shows that so far as digestion is concerned any of these periods may be used. Under-nourished infants require more food per unit of weight, and they may need more frequent feedings if the capacity of the stomach is small and the emptying time short. Well babies who take large amounts, who sleep well and are contented for the longer periods can be well managed on four-hour feedings. The food requirements based upon the nutrition and the body weight furnish a better means of estimating the total twenty-four-hour quantity than the hard and fast regulation of the volume fed at meals based upon the theoretical capacity for the age of the child.

**Physiology and Gastric Digestion.**—Gastric digestion of milk begins with the action of rennin which precipitates the casein. Casein then combines with the calcium to form calcium caseinate or paracasein. Bosworth has shown that the excess of calcium in cow's milk food interferes with the assimilation of fat and with the digestion and absorption of protein (casein). The action of rennin in curdling milk does not require the high degree of acidity needed for the action of the other gastric enzymes. Protein digestion is slight during the period that milk is in the stomach of the infant.

The amount of acid is normally much less in children than in adults, but after one year of age its production and concentration in the stomach increase with the age of the child. This is rapid up to the age of four, to remain constant until seven years. When the gastric contents of artificially fed infants are examined, there will not be enough free hydrochloric acid

found to be recognized. An increase in acid is dependent upon the kind of food which has been given, and upon the age of the child. This will be noticed especially in the feeding of sweet milk.

Test meals for use in studying the gastric acidity have not the importance in early childhood that exists in later life when ulcer and malignancy are common. Most studies on the gastric contents have been made in infancy following the feeding of breast milk. For determining the function a test meal consisting of from 4 to 7 ounces (120 to 200 c.c.) of equal parts of skimmed milk and oatmeal gruel is recommended by Mueller as the most satisfactory. The stomach contents are removed in one hour after feeding. Although the practical value of gastric analyses in childhood is small, there are many occasions when the determination of the gastric acidity might lead to useful suggestion for the feeding and therapy. Among these are hot-weather indigestion, atonic dyspepsia, persistent or recurrent vomiting, tetany, and celiac disease.

The relative acidity or alkalinity is spoken of as the hydrogen-ion concentration. This is expressed by the abbreviated  $P^H$ . A neutral reaction has a  $P^H$  of 7.0. As the acidity of a solution increases, the figure for  $P^H$  gets less, and is always under 7. Alkaline solutions are above 7.

The degree of acidity in the stomachs of normal breast infants has been found to average a  $P^H$  of 5.1. Enough hydrochloric acid should be secreted to bring the acidity of the contents to a concentration of  $P^H$  5.0 to 3.75.

*Causes Affecting the Gastric Secretion.*—Most secretion takes place in the first half hour after the food enters the stomach. There are numerous factors which influence it, chief of which are the presence and character of the food. An adequate amount of acid secretion in infants is found only in those who are breast-fed. As the baby gets older and takes more food the hydrochloric acid increases with the necessity for it. The good results from feeding buttermilk, albumin (protein) milk and any artificially soured milk are due to the somewhat predigested nature and the acid content of these foods whereby less gastric juice is required for their digestion. Undiluted whole sweet milk has always been known to be difficult of digestion by most infants. For many years this difficulty was ascribed to the large proportion of casein in cow's milk. Attempts were made to overcome the difficulty in feeding this artificial food by diluting with water or cereal by boiling, by adding alkali such as soda bicarbonate and citrate, lime water, and by numerous other methods. The explanation of the indigestibility of whole cow's milk lies in the demand which it makes of the acid secreting power of the stomach. The high content of salts (buffer substances) requires more acid for neutralizing them than is needed in breast milk. Therefore as explained by Marriott sweet milk may encounter too little acid for optimal digestion.

The stomach acidity in infants is definitely lowered during the digestive

period by elevated body temperature, whether due to external heat (hot weather or baths) or to infectious fever. Bacterial growth is more abundant in the gastric and intestinal contents when insufficient acid is present and the gastric secretion fails to destroy microorganisms which are thus allowed to reach the bowel and become active there. It is because of the decreased acidity during febrile attacks that food, especially cow's milk, must be reduced in amount.

There is a deficiency of the acid secretion in infants suffering from malnutrition. In infantile tetany, which is characterized by a marked reduction in the lime content of the blood, the acidity of the stomach is decreased and the emptying time shortened. Klementsson found acid reduced or absent (hypochylia or achylia) in chronic disturbances of the gastro-intestinal tract.

The acidity of the gastric contents in infants is said by Behrendt to be decreased by the feeding of heated breast milk. This is of practical significance as it concerns only the administration of drawn breast milk which has been dried, or has been boiled as a protection against the possible transmission of the spirochetes from a wet-nurse whose blood has not been proven negative. I have used such milk in the feeding of premature infants, but have seen no ill results, digestive or otherwise.

It is probable that many children with deficient appetites and with gastric pain have diminished secretions.

*Effect of Hunger.*—Gastric hunger is accompanied by contractions of the empty stomach, occurring sooner after meals in infants than in adults. These contractions have been noticed shortly after birth and before there has been any food. Hunger is greater in infancy and childhood, and contractions more active the younger the child. Carlson assigns this to the rapid rate of gastric secretion with quick digestion and a youthful and vigorous stomach which is more often empty. The infant gets hungry usually in two and one-half hours after a full meal.

In infants and young children the blood sugar decreases during hunger to a percentage under 0.07 per cent. In prolonged fasting it still further decreases. The longer the fasting the lower the sugar. This is the opposite of what occurs in adults. Nutritional disturbances in infants cause still more rapid fall in the blood-sugar content.

*Bacteria in the Stomach.*—The stomach of infants and children does not seem to be a favorable place for microorganisms. They are few in number and variety. It is generally considered that the action of the gastric juice is markedly bactericidal. The destruction of microorganisms and the active motility of the stomach prevent bacteria from developing and remaining therein. Two varieties may be said to be normally present: lactic and butyric acid bacilli. The butyric acid bacillus which is concerned in fermentation in both breast and bottle infants grows well in the stomach. Lactic acid bacilli are of little importance in infants and children. Staphylo-



cocci, streptococci, colon bacilli and fungi have been occasionally reported, but only rarely have they been pathogenic.

### VOMITING IN INFANCY

Vomiting during infancy is a common occurrence. Vomiting is only a symptom or expression of disturbed gastric function, the cause for which usually is outside of the stomach. It may be of little significance or it may be of the most urgent importance.

**Simple Regurgitation.**—A distinction should be made between the regurgitation of mouthfuls of food and the forcible expulsion of a considerable portion of the feeding. Infants nursing at the plentiful breast for a period of twenty minutes or more, or at too frequent intervals, will often give every evidence of thriving, while spitting up or regurgitating frequently. It is usually due to overflow or excess beyond the capacity of the stomach. The effect of gastric air in expanding or shifting from one region to another is to force out some of the liquid contents. The raising of the child suddenly from the recumbent to an upright position is sometimes accompanied by regurgitation, especially if the infant be grasped over the epigastrium. The condition need cause no alarm if the child is making a normal gain in weight.

The method of prevention is obvious. There should be reduction of too large an intake by shortening the nursing period and increasing the interval. The position of the child during and for one-half hour after nursing should be semi-erect, as any air swallowed is more easily brought up, with less tendency to expel liquids. The stomach empties more readily through the pylorus in this position.

There are many objectionable habits which facilitate regurgitation. Among these is the use of the pacifier which the infant constantly sucks. This causes a steady flow of saliva which is swallowed along with air from the nasopharynx. Another reprehensible custom is that of tossing, shaking and rocking the infant, causing splashing and regurgitating of gastric contents.

**Mechanical Vomiting.**—Every one has observed the ease with which vomiting occurs during inspection of the mouth by the application of a tongue blade or spoon. Too rapid intake may cause a momentary spasm of the esophagus or may permit the entrance of considerable air. The attempt of the stomach to contract about the contents will force out not only air but a portion of the liquids. Long nipples, whether human or artificial, may be drawn well back upon the tongue and produce gagging. The thumb, fingers or fist of the infant are often sucked and pushed far back into the mouth. Infants have the ability to reject food such as new articles of diet, especially when fed in a new way, or given quantities beyond their wishes. The breast baby may refuse to take bottle or spoon feedings and react by

vomiting. The bottle infant may throw up its food when attempt is made to feed at the breast or with a spoon. It is common to find that the change to a different formula, especially sour milk mixtures, will result in ejection of the feedings when they are first tried. It is easy to exceed the capacity of the stomach in artificial feeding.

Vomiting is apparently due in some cases to a lack of development in the centers governing sucking and swallowing. It may be due to deformities of the tongue, a long uvula, stenosis and foreign bodies in the esophagus. Obstruction in the duodenum or at the pylorus is a factor in the early days or weeks of life.

The peristalsis of any portion of the intestinal tract may be replaced by reversed peristalsis which is a forerunner of vomiting and of regurgitation of fecal material into the stomach, and found even as far up as the tongue. Fecal emesis is rare in childhood. Reverse peristalsis is also caused by hookworm infestation, causing attacks of recurrent vomiting. It may occur as a result of kinking at the junction of the duodenum and jejunum.

**Causes Inherent in the Food.**—The severe toxemias of pregnancy result in premature or dead infants in a majority of cases. Such premature infants born alive have a higher mortality than those with normal maternal conditions. Abt's figures give a mortality rate of about 25 per cent. Diffuse hemorrhages of the viscera are found at autopsy. An infant born of a toxemic mother may show toxic symptoms at birth. The ingestion of her milk may cause vomiting. The loss of food soon produces nutritional disturbance. It seems best to advise the prompt removal of a congenitally weak infant from the breast of the toxemic mother and to withhold the feeding of the mother's milk even to a healthy infant until the toxic symptoms have disappeared. When the mother recovers in a reasonable time, her supply of milk will return if nursing is followed regularly. Milk expressed from a healthy wet-nurse is the food best tolerated by the infant who has been injured in nursing the toxemic mother. The normal infant may be fed a simple mixture of cow's milk acidified by lactic culture or by the U.S.P. lactic acid with equal parts of boiled water and one or two teaspoonfuls of corn syrup or dextrimaltose to each feeding.

A superstition exists among the laity as to the danger of using milk which has been long retained in the breast. The mistaken notion is prevalent that after a period of days or weeks when the breasts have not been nursed, the milk becomes unfit for feeding.

Artificially fed babies seem to be tolerant of an infinite variety of food combinations, at least so far as the stomach is concerned. The most common fault is the excessive dilution of the mixture so that too great a quantity must be given if the appetite is to be satisfied. This may not only exceed the gastric capacity, but because of the thinness of the formula it is more easily expelled by the contractions of the stomach.

**Gastric Insufficiency (Atony of the Stomach).**—During infancy, loss of appetite and the occurrence of persistent vomiting may be due to an insufficiency of muscle tone, as a result of which the motility of the stomach is feeble and the emptying time delayed. If this occurs in the first two months of life the condition is suggestive of pyloric stenosis, but differs essentially in that the orifice is open, and there is no increase in peristaltic movements. Food, however, does not pass through the pylorus while the infant is recumbent. We are indebted to Linberg, of Sweden, for the recent clear description of this clinical entity.

As has already been mentioned, there are two muscular functions concerned with the emptying of the stomach. One consists of a tonic or grasping contraction by which the stomach wall adapts its shape to the food content. This has come to be known as the peristolic contraction. This grasping power is ineffective when the food is liquid. The other function is better known and consists of wave-like or peristaltic contractions from the cardiac to the pyloric ends, as well as contractions of the entire digestive tube. By this movement the food is pushed onward. In the atonic infant the gastric muscular efforts are either feeble or absent.

In later infancy the undernourished child may show a similar vice of stomach tone and motility, resulting in recurrent vomiting. It may be accompanied by a chronic inflammation of the mucosa and the accumulation of an excessive amount of gastric mucus.

*Symptoms.*—Gastric insufficiency in the newly born is shown when all feedings begin to be vomited as soon as milk is fed in any appreciable amount. The absence of appetite is also characteristic, and is due to the failure of the stomach to empty. Apathy, loss of weight and dehydration proceed with the vomiting and starvation. The normal but not increased peristaltic waves are present. Linberg found with the barium meal and fluoroscope that when the infant was raised to a vertical position the contents promptly passed through the pylorus. The efforts of the atonic wall are insufficient to force the food onward, but when assisted by gravity all feedings will pass through. These cases should be differentiated from pyloric stenosis, as operative treatment is contra-indicated.

*Treatment.*—In the course of one month, by persistence in the upright posture after nursing, the vomiting ceases and the stomach gains a normal tone as the general nutrition of the infant improves. The good effects are seen especially in the institutional infant who fails to thrive and who is generally atonic. When he is held up during the feeding and taken into the sunshine for an hour afterward, he vomits little and begins to gain in weight. Gastric lavage with warm water, or with 1 per cent soda bicarbonate solution, daily, is of marked advantage in the beginning of treatment, by washing out the excessive mucus and the retained débris.

**Eruption of Teeth.**—It must be admitted that there are infants who vomit at no other time, but may do so at the period of swollen gums from



dental eruption. It should be again emphasized that other physical causes should be searched for whenever vomiting occurs. A neuropathic tendency often coexists. Usually with so-called teething disturbance there is considerable peevishness and at night marked restlessness. In children who react easily to any abnormal state there may be some elevation of temperature. However, fever should be regarded with suspicion as due to some infection.

**Toxic Vomiting.**—Practically all infections and toxic states in infancy are initiated or accompanied by vomiting. Outside of the gastro-intestinal tract the most common are acute respiratory disease, otitis media, tonsillitis, the contagious and eruptive diseases, pyelitis and acute vulvovaginitis. The most significant and the primary causes are found in the gastro-intestinal tract. These are commonly acute indigestion, auto-intoxication, enteritis, peritonitis, inflammatory obstruction of the intestines and strangulated hernia. Hyperacidity of the stomach contents may provoke habitual vomiting. Congenital lues should be considered as a cause in those obscure cases which are cured by specific treatment. The good effect of mercurial inunction in this class of cases has been noted.

**So-called Intestinal Influenza.**—That pronounced gastro-intestinal disturbances do occur as epidemics in winter is a clinical fact. It is a matter of doubt whether vomiting and diarrhea are ever caused by direct intestinal infection with the influenza bacillus. It is more correct to regard the digestive upset as the result of parenteral infection, probably systemic in character with the upper respiratory tract as the point of entrance. A preliminary "cold," with or without fever, is the rule though the symptoms may have been overlooked. Digestive disturbance in such instances is probably toxic. When profuse nasal or bronchial discharges have been swallowed, or when paroxysms of coughing occur, vomiting may easily be produced.

**Vomiting of Central Origin.**—Acute poliomyelitis, epidemic encephalitis, intracranial hemorrhage of the newly born or accompanying accidental trauma of the head, tuberculous and syphilitic meningitis, hydrocephalus, and brain tumors are all accompanied by vomiting at the onset or during their course.

**Influence of Previously Frozen Milk.**—During freezing weather, the occurrence of vomiting lasting from two to seven days is found in young children fed cow's milk, but not occurring in breast babies. This has led to the opinion that the use of milk which has been frozen is responsible. The writer has seen repeated gastro-intestinal epidemics occur in severe cold weather. Pennington has found that physical changes do take place in freezing so that milk cannot be restored to the normal after thawing, with changes in the casein and lactalbumin through bacterial action. In the cases observed, only those known to have used frozen milk were included, though the milk may have been frozen before reaching the city distributor.



The symptoms are vomiting and because of the repetition and persistence, the loss of strength and weight may be marked. Fever and diarrhea may be present, but not necessarily so. The stools are usually small and few in number.

The treatment is frequent gastric lavage, in severe cases every four hours until the stomach is at rest. A laxative by the mouth should be given at the conclusion of the lavage. During the presence of such vomiting, no cow's milk should be used. Frozen milk should be well boiled if it is to be fed to young children.

**Treatment of Vomiting.**—In the experience of the writer, gastric lavage is indicated whenever vomiting is repeated. Prompt efforts should be made to find the cause, which should have its specific treatment. The most serious error is to overlook intestinal obstruction. Lavage will obviously be of no value in such cases, the treatment of which is entirely surgical and urgent. The dehydrating effect of persistent emesis is second only to watery diarrhea, and is equal if not greater than starvation due to other causes. After lavage the food should be withheld for twelve hours, but water should always be administered, and if not retained, it is to be given both by the bowel and under the skin.

Simple vomiting in undernourished infants is frequently stopped by the administration of concentrated milk-cereal mixtures. A commonly used formula is made by cooking a pint of fat-free or partly skimmed milk, a tablespoonful of sugar, and three or four level tablespoonfuls of farina, for an hour until the mixture is thick. From one to three ounces are given every four hours as the requirements and tolerance indicate. Later whole milk can be substituted for the skimmed milk of the mixture. Such food is fed with a spoon or through a large hole in a rubber nipple. Care should be taken to give plenty of boiled water two or three hours after these concentrated feedings.

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## CHAPTER IX

### NURSING AND ALIMENTARY DISTURBANCES IN INFANCY

**Nursing Difficulties.**—Caking of the breasts interferes with the quantity of the secretion, as a result of which the infant soon becomes dissatisfied. Withdrawal by expression of the milk is beneficial to the mother's comfort and maintains the supply. The problem that arises from the pain and infection of cracked nipples is a serious one for the mother and for the permanency of nursing. While the effort is being made to cure the nipples, the infant gets accustomed to bottle feedings. The combination of these influences usually results in drying up the breasts.

Infection of the breast is without menace to the child unless the pus reaches the milk stream. In puerperal fever, the milk will not transmit toxins or bacteria unless the mother is overwhelmed by the infection. However, the serious condition of the mother makes it advisable, for her comfort and welfare, to provide other nourishment for the infant.

The influence of maternal eclampsia upon the infant is injurious and may be rapidly fatal. I have been able to find one series of reported cases, that by Goodale, who observed three apparently healthy newly born infants die within a few hours after their first copious nursing from eclamptic mothers. The symptoms are much the same as eclampsia in the parent: chiefly abdominal distention, cyanosis, coma and convulsions. The milk must transmit toxins from the mother. In case the eclamptic birth results in a viable child, it is probably more or less toxic, and further injury may be transmitted through the milk. Such a child usually has albumin, casts or blood in the urine and may have convulsions. The infant born healthy will not be injured by nursing the mother when her toxic symptoms have disappeared. A small degree of albuminuria in the mother who has been eclamptic need not cause an interruption in nursing.

The infant discontinues the attempt to grasp flat or retracted nipples. Soon there is not enough milk at the orifice to stimulate the suckling reflex. Efforts to draw out the nipples are usually unsuccessful, and it is better for all concerned to put the infant upon the bottle.

Occasionally an infant is apparently "breast-shy." It will take hold of the bottle nipple, but cries when put to the breast. The cause may lie in a neuropathic constitution or in the absence of the suckling reflex in the mouth.

Difficulties in nursing are not confined to the breast. Rubber nipples cause considerable annoyance. Frequently one sees an infant who has tried for a half hour to get the milk out of the bottle. The nipple hole may be

too small, or it may become stopped by large flakes of overheated milk or cereal. Constant, intelligent effort is required to feed the baby successfully through a rubber nipple. The opening in the rubber is best enlarged by puncture from the underside with a red-hot needle.

Tongue-tie does not interfere with nursing. Observation of many cases has made me agree with von Reuss that they suckle normally either the breast or the bottle if no other abnormality be present. Some other cause than tongue-tie should be sought in the child who nurses poorly.

Snuffles (syphilitic rhinitis) causes the child to breathe through the mouth, which becomes dry. This renders the child uncomfortable, lessens the desire to nurse, and requires frequent interruption for breathing. The lips are sometimes cracked (rhagades), causing pain while nursing.

Acute colds in the nose likewise make suckling difficult through interference with ventilation. The child becomes tired from the increased effort and the time consumed in emptying the breast.

Stomatitis is always accompanied by pain in the effort of nursing, and when the inflammation of the tongue and mouth is severe, the infant may refuse to continue after a short trial.

In severe grades of amentia from cerebral paralysis, the infant often nurses or swallows poorly. Even more difficulty in swallowing is manifested when thick food is used. It may be necessary to feed the child with a tube indefinitely.

**Colic in Infancy.**—Colic is not a disease, but is in most cases an evidence of food intolerance or a neuropathic state. It is manifested by screaming when paroxysmal pain occurs in the abdomen. It is usually a manifestation of overfeeding of breast milk or of unsuitable artificial food; but in some infants so-called colic is really hunger, disappearing when there is a sufficient increase in the amount and digestibility of the feedings. Colic is sometimes mistaken for the acute pain of intestinal obstruction.

Those physicians who have practiced for years among infants have learned to recognize a well-defined nervous type in whom the feeding plays little part. It is often found that the parents are likewise "high-strung" individuals, or that other infants in the family were similarly affected. Colic is here a part of a symptom-complex, or neuropathic diathesis. These infants are usually on the breast, and the disturbances do not show improvement on changes in diet. This type of colic is described under the heading of gastro-enterospasm.

Infantile colic is a condition much discussed by the laity. A homely philosophy has grown up among grandmothers and old nurses who believe that certain infants are "born to be colicky" and that "three-months' colic" is a state as well-defined as infancy itself. Colic has always furnished much occasion for humorous lay references and for "jokes" in the newspapers, where the father is portrayed in his night clothes walking the floor with a crying child. The physician however cannot so regard persistent colic,

especially if it happens in his own family or in his practice, where he will find that patience becomes exhausted. One is often at his wits' end to find relief for the infant, the family and the medical adviser. In apartment houses, the disturbance caused by the frequently crying infant makes the situation intolerable.

The local changes in the intestine are not understood. It is conceded that pain is produced by pressure from gaseous distention. Circulatory disturbances are probably present in the intestinal wall. Colic takes its name from the earlier conception that it is due to a disturbance in the colon. The older textbooks gave much space to the symptoms and treatment, with little appreciation of the underlying or exciting factors. Many homely and nauseous remedies were recommended for its treatment.

Of much more practical value than a discussion of the mechanisms of such abdominal pain is the recognition of the general causes which produce colic. When these are corrected the painful paroxysms disappear.

*Manifestations.*—Colic occurs at any time during or between feedings, usually exaggerated by irregular or too frequent intervals. It may be intermittent, frequently repeated or continuous for an hour or more. When food is definitely undigested, pain occurs at any time day or night. At the onset the disturbance begins in the late afternoon and gradually lengthens until it persists till late at night. In some cases the stools show nothing abnormal, in some they are loose and undigested, and in others who are artificially fed there is evidence of fat indigestion with large, dry, hard, fecal masses.

Infants subject to colic are much disturbed in their rest and sleep, and the intestine contains increased gas which may be passed intermittently. The abdomen is much distended and the muscles held contracted. With the appearance of pain, the child screams, kicks and draws up the legs, throws its arms frantically, while the hands are clenched and the extremities cold. Some infants pull the hair, scratch the face, or grasp any accessible object such as the clothing. The face is congested and often covered with perspiration.

It is hard to mention colic without reference to the disturbed morale of the household. The mother becomes distracted with the persistent and recurrent crying of the child, and all the attendants are worn out and nervous. The mother finds that temporary relief is sometimes obtained by raising the infant over her shoulder and maintaining that position until belching occurs. She may be able to get the child comfortable by an enema which allows the flatus to escape.

The course depends upon appropriate treatment. Untreated cases may go on for months, and in some cases little relief until past the period of infancy. It should be again emphasized that colic is only a symptom, and that the underlying cause should receive the attention of the physician.

*Treatment.*—There can be no recognized therapy directed to the symptoms alone. Unscientific dosing with proprietary remedies, teas and opiates



will yield no permanent results, and have no place in the practice of the physician. For the temporary relief of colic, one should empty the stomach by lavage, the intestine by warm enemata. When one is sure from careful examination that appendicitis or other inflammatory states can be excluded, the child should be given one dose of castor oil. Hunger must be relieved by proper quantities of an easily digested food.

In general the treatment is the correction of the diet, and the use of antispasmodics, such as atropin. The dietary therapy is described under the heads of dyspepsia, constipation and gastro-enterospasm.

When immoderate or unexplainable screaming recurs in an infant it may be due to congenital syphilis. I have seen several such cases, in whom the crying was increased from local pain produced by movement or by pressure over the epiphyses of the long bones (Sisto's sign). Antisyphilitic treatment causes the disappearance of these symptoms.

**Digestive Disturbances in Early Infancy.**—In the first few days of life, after the disappearance of meconium, the stools become fecal from the presence of milk elements. These early fecal stools are different from the later movements when feeding has become well established. Their consistency is thin and mucous resulting from the intestinal irritation produced by bacterial fermentation of the high amount of sugar in the colostrum. This transient dyspepsia is a physiological rather than a pathological process. It is an evidence of a temporary or adaptive period in the digestion at this age. The transition from colostrum to the milk secreted later causes the stools to be somewhat less watery.

The first month of life is a critical period from the digestive and nutritional standpoint. It is not rare to find an infant, who during the first four weeks has gained little, if any, over the birth weight. From the fact that young infants may sleep at the expense of nursing, the failure to take sufficient breast milk may be overlooked. The opinion that breast nursing always takes care of the needs of the child is often misleading and causes a false sense of security. Breast milk is good as far as it goes, but in this day many women are incapable of producing enough. The types of artificial food, so greatly diluted and often unsuitable, are even more likely to result in a stationary weight during this first month of trial, and a primary or secondary digestive disturbance be produced. Insufficient or improper food is therefore responsible for beginning of a nutritional disorder which in this stage is unrecognized and may lead to serious consequences. The remedy lies in intelligent and experienced supervision of the infant during and after the newly born period. Infants who are not getting enough milk from their mothers should be fed small amounts of additional food in the newly born period. For this purpose, I have now used fresh lactic acid milk in more than five hundred cases during the first two weeks of life and find it has been uniformly well tolerated and preventive of nutritional losses.

**Starvation Fever in the Newly Born.**—The occurrence manifests itself by a sharp rise of temperature to  $103^{\circ}$  F. or higher, on the third or fourth day of age. It is found in either the premature or full-term infant in whom underfeeding is common until the breast milk is furnished in sufficient amounts, usually up to the fifth day. The infant usually gets little fluid. As the days pass, the infant draws upon its high water reserve in the tissues, and this becomes definitely depleted as shown by the loss of turgor and weight in the first few days.

The clinical syndrome appears at a time when the meconium stools have not as yet been entirely replaced by milk feces. The microbic invasion of the contents begins at birth and the change in the character of the intestinal microörganisms has been offered as an explanation of this fever. Morse believes that there occurs a putrefaction of the meconium or intestinal contents, and he has designated it as intestinal toxemia, or intoxication of the newly born. He felt that such a contention is borne out by the curative effect of a dose of castor oil. Other writers believe that protein or bacterial products absorbed at this time by the intestine cause intoxication.

While a certain amount of water depletion undoubtedly occurs in all newly born infants, not all develop fever. Therefore it cannot be regarded as certain that a deficient intake of water or food explains the clinical symptoms (Grulee and Bonar). The fact that increased food or fluid results in a prompt cure is definitely known.

In differentiating starvation fever from other febrile disturbances one should remember that in the premature child, especially, the heat-regulating mechanism is unstable, and sudden elevations are easily produced by heat from water bottles or when the temperature of the incubator is too high. I have seen a premature infant attain a fever of  $105^{\circ}$  F. when inadvertently the temperature within the incubator was allowed to reach a similar height.

In case artificial food has been begun when this febrile rise occurs a digestive upset might be supposed, but vomiting and undigested stools are not found in this condition.

In order to be sure that the fever is of this type, all infections must be ruled out, especially otitis media, rhinitis, suppuration of the umbilical cord, erysipelas and pyelitis. The urine should be examined for microörganisms and pus. Intracranial hemorrhage from birth trauma is to be thought of, but its characteristic symptoms are missing.

*Treatment.*—In all newly born infants plenty of water should be given as a routine. A rise of temperature should be the occasion for seeing that the child actually swallows from 1 to 2 ounces of fluids every two hours. It is easily determined whether the infant is getting any food from the breast or how much it is taking from the bottle. From 1 to 3 ounces of a milk formula are to be given every three or four hours while awaiting the supply from the breast.

**Heat Exhaustion in Infancy.**—Thermic fever is responsible for much sickness among infants during hot weather when the digestive processes are greatly inhibited. Heat exhaustion is more apt to occur in poorly nourished or sick infants to whom insufficient amounts of water are given, or in whose surroundings the conditions are unfavorable for the maintenance of comfort and well-being. There is a direct relation between the excessive amount of clothing worn by babies and the occurrence of intestinal disturbances and high mortality during the hot weather.

The disturbance is most common in extremely heated terms when for days and nights there is no relief from the superheated air and the high humidity. The infant develops a sudden elevation of temperature from 103 to 110° F. There occur complete loss of appetite, vomiting, dryness of the skin, a rapid and feeble pulse. In severe cases there may be insufficient time for the development of intestinal disturbances, and the child's condition rapidly becomes worse with the appearance of shock, convulsions and coma. In fatal cases the end comes within twenty-four to forty-eight hours.

Prophylaxis is more effective than any treatment. Infants in hot cities have had little done for them in making possible the mitigation of intolerable heat. No clothing should be worn, and the child should be kept quiet in the coolest surroundings possible, preferably in the shady yard. It is better to reduce the strength of the food by a larger proportion of diluent.

Either as a preventive or when thermic fever occurs, the sponging of the body and cold applications to the head should be repeatedly used. Water should be given almost constantly, and the hypodermic use of a pint or more is the most useful therapeutic measure. Cases that have symptoms of shock and a subnormal temperature require stimulation by hot drinks such as tea, warm applications to the body, and the hypodermic administration of a few drops of adrenalin.

**Alimentary Anemia in Infancy.**—Marked degrees of anemia are not infrequently seen in infants who have been underfed for several months. Although this proves to be of the secondary type, the hemoglobin content may be so low that one's first impression is that he has to deal with a leukemia or other primary form. It may occur in the breast-fed child who is insufficiently nourished. It is more common with bottle feeding of proprietary formula and in mixtures made up of high fat and small amounts of the mineral salts, when fruit and vegetable juices are not added.

It is also generally accepted that the feeding of an exclusive milk dietary over an extended period will produce anemia in older infants. This usually begins between the seventh and twelfth months. The cause is an iron starvation. The retention resulting from artificial foods is not so large as occurs from the feeding of adequate amounts of breast milk, in spite of the fact that breast milk has a lower mineral content. Infants who have never had breast milk and the good start which maternal nursing gives

are more susceptible to anemia. Frail infants, especially the premature and syphilitic, may begin life with low hemoglobin. So it is probable that other causes in addition to the type of feeding may be responsible. The retention of food elements by the body is just as important as their presence in the formula. Certainly the same diet does not produce anemia in all children.

It has been my impression that the prolonged exclusive feeding of goat's milk is even more provocative of anemia. A few references have appeared in the literature which bear out this assertion. Daniels and Stearns studied the relative retention of the food elements necessary for growth with goat's milk as compared with cow's milk. Much less nitrogen was retained when goat's milk was fed. Brouwer found clinical as well as hematological evidence of anemia during the first year of life when goat's milk was used more than a month.

The treatment with cod-liver oil, organic or inorganic iron, and arsenic raises the hemoglobin of the blood. It is well to use medicinal iron as well as iron-containing food, as the milk diet is deficient in this mineral, and the clinical improvement slow. The infant should be given the benefit of outdoor sunshine and freedom from infection. Above all, the diet should be changed, preferably to soured milk and a more general variety. The yolk of egg, prune pulp, puree of peas or beans should be fed for their high iron content.

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## CHAPTER X

### DISEASES OF THE STOMACH

Organic disease of the stomach is uncommon in childhood except as it accompanies or is secondary to diseases in the intestine. The only constant clinical manifestation is vomiting, a symptom, however, which results from such a number of causes that it is not correct to assign it to local gastric disease except when pyloric obstruction, ulcer, dilatation or irritant poisons are factors. Gastro-enteritis is a term often carelessly employed, as the involvement of the stomach, other than in a functional way, can seldom be known except at autopsy. When the gastro-intestinal tube is opened at postmortem, the stomach generally shows no evidence of inflammatory changes, but the gastric wall often presents more or less discoloration and softening which have taken place after death. Many years ago John Hunter taught that there are few dead bodies in which the stomach is not to some degree digested by the gastric juice.

With the exception of the young infant, most gastric disturbances are functional or symptomatic. In the older books on children's diseases, there was considerable space given to indigestion and vomiting, both of which were considered as diseases and not appreciated as symptoms. In our present state of knowledge, which is far from complete, but which has grown considerably by reason of the roentgen ray, bacteriological and autopsy examination, it is considered that with few exceptions pathological lesions in the gastric wall seldom occur. The slight vulnerability of the stomach is in marked contrast to the intestine, in that there is a high degree of immunity to bacterial invasion of the gastric structures. Occasionally it may be involved secondarily in a diphtheritic or streptococcic infection found at autopsy to be a membranous gastritis. It has been found rarely to share in a membrane formation as an extension from an enteritis.

### ACUTE GASTRITIS

**Catarrhal Gastritis.**—This affection is rare except as an accompaniment of diarrheal disease of childhood. The pathological changes are slight, seldom more than congestion and superficial inflammation of the mucous lining of the stomach. At autopsy one may find a denuding of the epithelial surface, but it must be remembered that death is not due to any lesions of this nature, as catarrhal gastritis is never severe enough to prove fatal. It plays no major part in the serious intestinal disease with which it is associated.

Clinically, acute catarrhal gastritis is similar to acute gastric indigestion. The latter must be regarded as a functional disturbance without pronounced pathological or inflammatory changes. In this category must be placed those simple short attacks of gastric intolerance which occur in children who partake of some food which is vomited in the course of a few hours. Certain foods cause allergic symptoms in the sensitized child. Spoiled food such as milk, ice cream, and canned fruits, meat or vegetables, produce the disturbances by their poisonous or toxic content which is now recognized as contamination with the paratyphoid or botulism organisms.

**Symptoms.**—It is difficult to ascribe a distinct symptomatology to catarrhal gastritis, for the symptoms are combined with those of the intestinal and constitutional disturbances. Vomiting is the most common manifestation. If there is definite inflammation of the stomach mucosa, vomiting will persist until the gastric injury subsides. No water or food is retained. Nausea and retching result in the evacuation of brownish or bile-stained mucus, food remnants, and finally traces of blood. As a result of the fluid losses, the child is intensely thirsty and will drink all water offered, but vomits it promptly.

The child's discomfort is marked, due to nausea and the persistent involuntary contractions of the irritated stomach wall. In older children pain is complained of in the epigastric region, which may be tender on palpation. After prolonged vomiting the abdominal muscles may become tender from the excessive efforts.

The general symptoms are loss of appetite, prostration and fever. In the beginning there is a marked rise of temperature, but fever subsides as soon as the vomiting or associated intestinal disturbance has disappeared, which is usually in from three to five days.

The other manifestations are a coating of the tongue, not infrequently a stomatitis, and a foul breath. Usually there are diarrheal stools, but constipation may be present in older children.

If feeding is resumed too early and the character of the food is not simple, the disturbance may continue for a week or ten days. Relapses are not uncommon in the delicate child after apparent convalescence.

**Treatment.**—The treatment should be begun with the cessation of all attempts at feeding. Vomiting is reduced by the repeated lavage of the stomach. Cool water, to which is added a tablet of Ringer's formula, is used for washing out the stomach, and repeated twice daily until the stomach no longer shows unrest. A mild counterirritant, such as a mustard-flour paste, is of help when applied over the epigastrium for a few minutes every eight hours, carefully avoiding blistering of the skin. Bathing, rest in bed, and all measures which increase the personal comfort are to be recommended. The bowels should be irrigated once daily during the acute stage, leaving some of the fluid in the bowel to be absorbed. When vomiting is stopped, and the appetite returns, suitable food should be offered the

child. In the breast-fed infant, nursing can be at once resumed. In the bottle-fed child, milk is not begun until the stools are normal. Meat, broth, gruel, gelatin, fruit juices and toast are foods which digest easily and are useful in the child past one year of age. Small amounts are advisable until tolerance is reëstablished.

If the digestive disturbance is manifestly one of intestinal origin, the dietary treatment must be arranged accordingly. This is discussed elsewhere in this volume.

### ACUTE TOXIC GASTRITIS

This is the most common and serious of inflammations of the stomach in children. It is produced accidentally by the swallowing of caustic substances. The poisons to which children are commonly exposed in the household are concentrated lye and cleansing powders, carbolic acid, ammonia, camphor and bichlorid of mercury. Phosphorus-containing fireworks, especially "spit-devils," are also swallowed by young children.

**Pathological Lesions.**—The gastric changes from the action of corrosive poisons are not different in childhood from those in later life. They will be severe or even fatal depending upon the quantity which reaches the stomach, especially if the stomach is previously empty. The local effect is the production of intense inflammation of the mucosa or entire wall, with multiple ulcerations, hemorrhage, perforation and peritonitis. Death may be due to the corrosive injury or to toxic absorption.

**Symptoms.**—The swallowing of caustic alkali and acid substances causes pain and inflammation of the mouth, tongue, pharynx and esophagus. Spasm of the esophagus is usually produced so that vomiting promptly occurs. As soon as the poison reaches the stomach, there is further emesis, and the vomitus is often stained with blood. There is marked gastric distress or pain. In spite of the thirst the child is unable to swallow. The poison may cause collapse and death within a few hours or days. Gastric and intestinal inflammation result in those cases that survive the initial shock. Infants and young children usually succumb to the effects of the poison. Milder degrees of injury, due to less amounts of irritant ingested or to neutralization by antidotes always leave cicatricial deformities in the wall of the stomach.

**Treatment.**—Most efforts at treatment are too late to be of benefit. It is fortunate if only a small amount of the irritant has been swallowed and if the stomach contains some food or water. It is well if the mother knows that the best remedy is that which is near at hand, and gives the child all the milk, egg white and mineral oil that can be swallowed. Unfortunately the child is often unable to swallow, and the only hope is in prompt vomiting.

On the arrival of the physician, a soft stomach tube should be gently passed, and demulcents given, the stomach washed out, and more fluids

introduced. The child should have relief from pain by a hypodermic of codein or morphin.

The physician should have at hand the information as to the best antidotes. He should use substances which are incapable of causing further irritation to the stomach or of jeopardizing the safety of the child.

Antidotes are administered with the object of forming an insoluble compound which renders the poison inert, or of diluting or covering the poison so as to interfere with its absorption. As soon as the antidote is given, the stomach should be washed out, or an emetic given. The antidote should be something which is readily obtainable, such as egg for mercury, liquid petrolatum or melted vaselin for phosphorus, vinegar or lemon juice for alkalies, baking soda or soapsuds for acids. Large quantities of water are to be added to all antidotes, and repeated for twenty-four hours so as to increase elimination by the kidney.

## CHRONIC GASTRIC INDIGESTION

The existence of an irritable stomach, the result of congenital weakness, long-continued improper feeding, neuropathic states in the child, and severe nutritional disturbances is not infrequently observed. That it is due to a chronic catarrhal gastritis is difficult to prove. Repeated attacks of acute gastritis may result in degeneration of the epithelial lining of the gastric glands. It is more probable that the musculature of the stomach wall becomes atonic as a result of repeated infections and nutritional diseases. Dilatation of the stomach usually develops, a condition which interferes with the motility and emptying of food, whereby fermentation and excessive amounts of mucus are produced. From all these causes gastric digestion is impaired. Gastropsis interferes with the functions of the organ and is present in many children after the age of three to five years.

The symptoms referable to the stomach are few. The affected child has a variable appetite, usually poor, but sometimes immoderate for inappropriate articles of diet. The breath is offensive, and the tongue may be heavily coated. The stomach is distended. Beyond these symptoms the remainder of the clinical picture is one of a poorly nourished individual with thin abdominal muscles, prominent abdomen and poor general body tone. There is usually constipation, though attacks of diarrhea are easily excited when a poorly functioning stomach is overloaded and food is incompletely prepared for the intestine. Chronic ill health is evident. The child is irritable, sleeps poorly, and either cannot be made to gain in weight, or loses steadily and suffers from anemia. There is often albumin in the urine due to the effect of poor body posture.

**Treatment.**—A radical change is needed in the environment and routine of the child's life. This is more important than any medicinal treatment, although the administration of dilute hydrochloric acid, strychnin, iron and



pepsin is of some benefit. The child should have twelve hours of sleep at night and a midday rest of two hours. Change to the country is desirable, where there will be plenty of fresh air, sunshine and freedom from excitement.

Three meals daily of easily digested and appetizing food are advisable. Sweetmeats and highly seasoned articles should be avoided. A pint of milk in twenty-four hours and plain well-cooked food will be handled better than special diets of which the child easily tires. In older children it is of great assistance to elicit their coöperation and to stimulate their pride in the effort to regain health and weight.

In addition to the hygienic and dietary régime just suggested, much benefit to the gastric function and the relief of intestinal stasis are obtained from the wearing of a proper abdominal corset belt. The results from persistent treatment are good. When individuals pass into adult life with poor digestion, it is evident that their earlier health and nutrition have been neglected, or that severe acute or chronic general infections have interrupted the normal development.

### DILATATION OF THE STOMACH

Chronic dilatation of the stomach occurs from the muscular atony in rickets, in celiac disease, and in pyloric and duodenal stenoses.

Acute dilatation of the stomach results from toxic states and from over-feeding. It is liable to happen to the infant in a severe marantic state when too much food is given.

It may occur in severe upper respiratory disease, especially pneumonia. In such weakened states, distention may cause collapse and embarrassment of the respiration.

The diagnosis of dilatation is made by the appearance of a gastric fullness which can be seen in the epigastric region. The barium meal and radiogram clearly show the dilatation. Percussion gives an abnormal area of tympany, reaching below the umbilicus. The dilated stomach holds large quantities of fluid. Vomiting occurs daily and brings a larger amount of contents than normal for the child. Frequently dilatation of the stomach is unexpectedly found at autopsy.

The treatment is gastric lavage and withdrawal of gastric contents once daily with the catheter. Concentrated or small amounts of food should be fed rather than usual amounts.

### HEMORRHAGE

When the child vomits blood the question of first importance is the source, and whether bleeding is actually occurring in the stomach. In a nursing infant blood may be swallowed from the mother's cracked and

bleeding nipples. Blood from epistaxis may be swallowed. After tonsillectomy the stomach at first contains much blood, and the contents should be later watched for evidence of continued bleeding.

Hemorrhage from the stomach is of little significance or seriousness so far as local disease is concerned. The most common appearance is due to persistent or immoderate retching, whereby the vomitus shows streaks of bright blood, or contains some dark brown partly digested bloody material. Not all the blood is vomited, some passing by the bowel.

In the newly born, so-called melena is the most common variety of bleeding, usually shown by bloody stools and also by bloody or dark vomitus. The term melena means not only hemorrhage from the bowel, but also black vomiting. The term is only a symptomatic one, and should be dropped, as we now know that gastro-intestinal bleeding in early days of life is due either to a hemorrhagic diathesis (not hemophilia) or to gastric or duodenal ulcer. The blood in the true hemorrhagic disease comes from the stomach or esophageal wall. The diagnosis of this type of bleeding is made by a simple estimation of the bleeding and coagulation time (see Rodda or Duke). Blood which has not clotted after ten minutes is definitely abnormal, and the child may be regarded as a bleeder if more than ten minutes are required for oozing to stop. Successful treatment is by the subcutaneous or intramuscular injection of whole blood obtained from the parents. The amount should be 20 to 30 c.c. and in serious cases as much as  $\frac{1}{60}$  of the body weight. Such a large amount must be given by transfusion.

Bleeding as a result of ulcer of the stomach is rare (see ulcer) past the age of the newly born.

## ULCER OF THE STOMACH

Cases of acute and chronic gastric ulcer in children, while rare, are being more frequently reported. Most writers on the subject have stated that ulcers are seldom discovered except at autopsy. In recent years they are beginning to be found not only accidentally during an operation, but a preoperative diagnosis is occasionally possible. Clinically gastric and duodenal ulcers are usually classed under one heading. Most of the symptoms are similar. Peptic ulcer also occurs in Meckel's diverticulum during childhood (see diverticulum).

**Occurrence.**—Few physicians have ever recognized a case of gastric ulcer, either because of the rarity in childhood, or from the fact that little attention has until recently been paid to the diagnosis at this time of life. Fischl regards the infrequency in infancy as due to the small amount of gastric acid secreted, the rapidity with which the infant empties the stomach, and the fact that mechanical disturbances of the stomach are seldom present in the infant. The nature of the food (consisting entirely of milk) may have much to do with the prevention of pure peptic ulcer, as the

acid of the gastric juice is bound by the buffer salts of the milk. Proctor has recently found in the literature only nineteen cases of true chronic ulcer in children, in some of whom the location was in the duodenum. Of 1600 gastric ulcers in a large clinic he reports only one in a child. The general opinion is that ulcers are less rare in infancy than in later childhood. In adults it is not infrequent for the symptoms to date back to childhood.

Holt found multiple ulcers in six of 390 routine autopsies. One observer found 7.55 per cent of 172 cases were in children. In the reported cases they are apt to be found in three general periods of childhood. The newly born infant is relatively the most susceptible, and manifests symptoms of obscure vomiting or of hemorrhage from the bowel which is regarded as melena. In later infancy ulcer may be an accompaniment of marasmus. After the age of five years chronic round ulcer is the most frequently reported.

**Causes.**—Ulcer and intestinal hemorrhage in the newly born have been ascribed to the change occurring in the circulation at birth, or to the effect of toxemia and prolonged labor. Spontaneous intestinal hemorrhage does occur in some newly born infants, but not all of these have bleeding from ulcerated surfaces. It is only at this time of life, however, that ulceration may be accompanied by a prolongation of the bleeding and coagulation time, but ulcer in the newly born is usually a primary manifestation.

Högler found ulceration present in all cases of melena coming to autopsy. The ulcerations are not limited, however, to the stomach. Blood in the stools of the newly born is probably more common than heretofore suspected. It may be masked by the meconium. Högler concluded that occult blood will be found in the stools of 50 per cent of the newly born, and that gross bleeding occurs in one of every 500 births.

After the first month, ulcer is a secondary manifestation, and occurs as a result of acute or chronic infection, metabolic disturbance, nephritis, uremia, burns, eczema, trauma and circulatory disturbances.

In older infants suffering from marasmus, ulcer may result from the reduced amount of blood in the circulation. The theory of Rosenow is that chronic ulcer results from infection coming from acute coryza or tonsillitis.

**Pathological Lesions.**—In certain cases the lesions are inflammatory, and in others they represent thrombosis in the vessels of the mucosa. In the infant there is usually more than one ulcer, confined to the mucous membrane. They are most severe in the posterior wall of the stomach near the pylorus.

The chronic peptic ulcer is from 1 to 3.5 cm. in diameter. Perforation occurs in long-continued cases, opening upon a neighboring viscus such as the spleen, pancreas or liver, and resulting in local or general peritonitis. Stenosis of the pylorus is apt to occur when the ulcer is in that region. The ulcer is considered chronic when it is known to have lasted as much as two

months or when it is indurated and has elevated borders, and has become adherent to adjacent structures.

**Symptoms.**—Ulcer appears with manifestation of hemorrhage in certain cases, and results in sudden death. In the chronic form the symptoms are indefinite but consist chiefly of vomiting and either occult or evident blood in the stools. The possibility of ulcer should be considered in any infant suffering from marasmus and in children who have suffered a severe burn.

In later childhood the symptoms are more characteristic. There is undoubtedly a history of obscure chronic or recurrent digestive disturbance, though this may be overlooked by the family. The child may have been well for certain intervals, and at others complain of pain or distress in the region of the epigastrium or elsewhere in the abdomen. The character of the pain is much the same as found in adults. Food relieves the distress temporarily, as does vomiting which occurs soon after eating. The pain may appear at any time, day or night.

The general condition of the child suggests a long-continued derangement of the digestion and nutrition. There may be impairment of the appetite, progressive loss in weight and failure in growth, and there is usually constipation and in some cases tarry stools. The examination of a test meal is unsatisfactory, as hyperacidity is not present. A roentgenogram should be made and although unconvincing for ulcer it may suggest some abnormality. When blood is found in the vomitus or stool of these cases the diagnosis is fairly certain. The coagulation time is normal. When blood is not present, the diagnosis is difficult. Perforation is a late manifestation, but it may be the first to call the attention to the fact that the disease is located in the stomach. Peritonitis and death result promptly after acute perforation. The disease is apt to be mistaken for appendicitis.

**Treatment.**—The writer personally has not known of cases operated upon in infancy, but the literature contains a few references to successful surgical intervention. Operation in time to save the life is possible and these children will react as well as adults. The ulcer should be excised and if pyloric obstruction be present the suitable surgical treatment should be carried out.

It is now the rule to give an intravenous or intramuscular injection of blood to all children who suffer a severe intestinal hemorrhage.

The dietetic and medicinal treatment of chronic peptic ulcer is similar to that in later life.

## FOREIGN BODIES IN THE STOMACH

The variety or number of foreign bodies which reach the stomach of children is limitless. As soon as the infant can grasp accessible objects they are at once carried to the lips and incredibly large ones may be swallowed. The tendency persists through the runabout and to a less extent in the



pre-school age. It is not rare to find the school child who puts coins or marbles in the mouth. Foreign bodies are swallowed voluntarily or accidentally.

It is very common for the family to be mistaken as to the fate of some object which the child has been seen to put into the mouth, and which may be lost rather than swallowed. Occasionally a foreign body may be vomited, or one found accidentally during an x-ray examination. Mothers frequently ascribed illness and fever to ingestion of these objects. Symptoms are not probable as such objects usually cause no discomfort and certainly no pathological processes. A rare exception to this was reported by Cuadra in the case of a three-months-old infant who had diarrhea and fever ascribed to the ingestion of a bird feather, which was discovered in the stool. In older children, I have never heard of such symptoms occurring.

It is likely that ground glass will provoke gastric bleeding, but I have known the swallowing of small particles of glass to cause no symptoms whatever. In watching the progress of an open safety pin through the gastro-intestinal tract by daily radiograms, no pain or hemorrhage occurred at any time. On the second day the pin was out of the stomach and on the fourth day it was well down in the intestine. In spite of constant effort to discover the pin in the stool, it was passed without any discomfort to the child, and without the knowledge of the mother and trained nurse. Usually more time is required for the passage of a foreign body than is taken for food, and the period will vary with the shape and size of the object.

Recently I saw, by the radiogram, the presence of a buckle in the young child's stomach. It did not pass the pylorus for several weeks. One surgeon advised its operative removal. The family was urged to let it alone, and the buckle was recovered from the stool two months after the accident.

Occasionally the foreign body is regurgitated into the esophagus where it may become lodged, and require removal by the aid of the esophagoscope.

Nervous children may pull the hair and swallow portions which may be the starting of hair-ball in the stomach or of an intestinal fecolith. Hair-ball is usually not discovered until later in life, usually at operation upon the stomach for some other cause. Children will pick up threads and strings from the floor, shreds of fur and lint from clothing or blankets and put them in the mouth. The presence of these in the pharynx or esophagus may cause vomiting.

**Treatment.**—Unless there are marked local symptoms such as suffering or penetration, both of which are rare, foreign bodies in the stomach should be left alone as they show a tendency to pass spontaneously, even if of large size. The child should not be given laxatives, but the usual diet should be maintained and the foreign body watched for. It may take months to pass. I have never known of a gastrotomy being necessary for any object which reaches the stomach.

## MALFORMATIONS AND TUMORS

With the exception of hypertrophy of the pylorus, malformations of the stomach are rare. Hour-glass stomach has been reported. The stomach may be out of place, transposed to the right side of the abdomen, to the chest cavity through a hernial opening in the diaphragm, or to the lower abdomen due to ptosis.

Benign or malignant tumors of the gastric wall are so uncommon as to have escaped the observation of most clinicians.

## HYPERTROPHIC PYLORIC STENOSIS

**Descriptive Summary.**—Symptoms usually appear during the first six weeks of life. The affected infant begins to have projectile vomiting shortly after each feeding. Soon the stools become small or infrequent. It will be noticed that the change from breast to numerous other foods gives no relief. When the abdomen is carefully watched, patterns of the gastric contractions will be seen as peristaltic waves rhythmically crossing the upper abdominal region. A small tumor may be felt near the median line in the upper right quadrant, most noticeable during the peristaltic wave. The infant gives marked evidence of hunger. The nutrition steadily fails, going on to emaciation. In spite of the vomiting of large quantities of food and mucus, a considerable residue may be siphoned off through a catheter. Under the fluoroscope, little or none of the opaque meal will be seen to pass the stomach. Surgical exploration reveals an obstructive tumor at the pylorus.

**Nomenclature and History.**—Hypertrophic pyloric stenosis is now the accepted term. Many writers prefix the word “congenital.” In Germany the name “Pylorospasmus” is used in preference to others. Much discussion has occurred in all countries as to the identity of pylorospasm and hypertrophy, as a result of which both terms may be found in the literature. In America what difference of opinion there may be is mostly among pediatricians, the surgeons holding the conviction that the disease is definitely an hypertrophic manifestation. Laying aside the mooted points as to the congenital nature and as to whether tumor formation is a later stage of spasm, the nomenclature should be based upon the finding of a physical obstruction at the pylorus where at operation a tumor is invariably found. The writer prefers to consider pylorospasm as a separate entity under the title of gastro-enterospasm.

Probably the first cases ever reported were those by George Armstrong in England, in 1777, under the title “Spasm of the Pylorus,” and Hezekiah Beardsley in Connecticut, in 1778, under the subject of “Case of Scirrhus of the Pylorus in an Infant.” The reader is referred to the excellent his-

torical review by John Foote. Only a few other cases are found reported in the century that followed. The clinical symptoms and pathological findings were well described by Hirschsprung in 1888 and by Ibrahim in 1905 and later. Since then the literature has been full of reported individual cases and series of cases, and the treatment, both medical and surgical, has been highly developed. The disease is still comparatively unfamiliar to those practitioners who have given but little study to infancy.

**Etiology.**—It has been my experience that the disease has often been undiagnosed because of the custom of ascribing vomiting in early infancy to a child's supposed lack of tolerance for breast milk. The history of the disturbance will here be of value. It is usual for the mother to emphasize the fact of the vigor and satisfactory nursing of the infant during the first days or weeks of life, when suddenly without apparent reason a whole feeding will be forcibly ejected, and the vomiting become a constant occurrence. No influence of the breast milk *per se* can be assigned to the development of hypertrophied pylorus. The fact that breast milk is being regularly vomited and in a forcible manner should at once suggest pyloric obstruction.

Hirschsprung regarded the disease as primarily a muscular hypertrophy. An hypertrophic pylorus has been found in a fetus. He concluded that the tumor must have been primary, as a spasm of the pylorus would be improbable in fetal life. If the growth were always congenital the freedom from symptoms during the early weeks of life would be explained only on the basis of incomplete obstruction during that period, with final enlargement of the musculature to the point of causing symptoms. Others have offered the explanation that superimposed upon the primary hypertrophy there is a secondary spastic contraction of the muscular coat, which in certain cases relaxes at times so that food will pass. Pfaundler's theory was that the spasm is primary and the thickening is compensatory from the narrowing of the pyloric canal. Another explanation is that of Heubner ascribing the development of obstruction to a pre-existing spasm of the entire stomach wall. Numerous observations have shown that the hypertrophy persists for months after spontaneous cure of the vomiting. There are so many causes for vomiting in early infancy that one must insist upon the presence of classical symptoms of pyloric obstruction before any conclusions in a given case can be drawn.

It is evident from the previous brief outline of the theories advanced that no explanation is as yet convincing. We are left to the conclusion that in the early weeks of life there is a predisposition to the development of hypertrophy of the pylorus, and that the vomiting and other symptoms are caused by the obstruction of the pyloric canal.

**Incidence.**—Textbooks of ten years ago considered it rare. It is impossible to estimate the case incidence. The pediatrician may have two or three cases at one time, or he may not see any for six months. The busy

surgeon is not infrequently called upon to operate, and series of twenty-five or more are not rare in reports of writers upon the subject.

Boys are definitely more commonly affected than girls. In fifty-two cases, Herz found only eight among girls.

Numerous instances of more than one member of the family affected by the disease have been reported. The writer had two cases in one family and three in another. Twins may be similarly affected. So far as is known there is regularly no familial factor in the etiology.

**Physiology and Pathology.**—There is a marked increase in the peristaltic function of the stomach. In spite of the increased contractions, the gastric contents cannot be made to pass the pylorus. According to Alvarez, reverse peristalsis is present in pyloric stenosis. There are also other causes for the vomiting. The attempt of the stomach wall to contract down upon the contents forces out liquids and the only way they can go is through the esophagus. Toxemia may be a secondary factor in the production of vomiting.

The stomach is greatly dilated, its walls thickened, and the weight of the retained contents causes sagging which still further interferes with gastric emptying by producing a kink at the pyloric antrum. The pyloric tumor is made up of greatly thickened circular muscle tissue, composed of the unstriped muscle cells, but there is no increase in the connective-tissue fibers. In gross appearance the tumor resembles hyaline tissue. The lumen of the pylorus is much reduced, and may be so completely obstructed that water will not pass.

There has been some discussion as to the fate of the tumor following treatment. The most convincing statements are those of Wollstein. After operation with division of the circular fibers she found that the wound in the pylorus healed in about two weeks. Early healing is brought about by the growth of the serous and submucous layers. Two years later there is only a connective tissue line to mark the site of the wound.

In cases getting well without operation much longer time is required for the tumor to disappear.

**Symptoms.**—It is noteworthy that pyloric stenosis is found in the breast-fed infant usually, with a previous history of normal digestion and development. Vomiting suddenly begins at the age of from one to six weeks, seldom later.<sup>1</sup> The infant does not vomit repeatedly between feedings, but usually once after each nursing, or several meals may accumulate and then be expelled.

In the beginning the child does not seem acutely ill; there is little or no elevation of temperature, the appetite is usually good or even increased. At this time of life it should be borne in mind that projectile vomiting is due to an obstructive origin. Gastric intolerance in a previously normal infant

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<sup>1</sup> In cases collected by Sauer, it is shown that 75 per cent began in the first three weeks of life.



without evidence of overfeeding or of intestinal indigestion furnishes one of the outstanding symptoms. The quantity vomited may be greater than the amount just taken. This is due to the accumulation of mucus and the retention of considerable portions of previous feedings.

There are two methods of estimating the degree of pyloric obstruction and the abnormal gastric retention. Both are useful. One of these is to measure the amount of contents which can be siphoned from the stomach four hours after the feeding. In the normal infant there should be no residue at that time. In pyloric stenosis, much food remains. Withdrawal of any considerable amount is evidence of abnormality in the emptying time. After this conclusion has been reached, it may be of help to secure the evidence of the fluoroscope or radiogram. Immediately after giving barium suspended in buttermilk, the child is held upright against the screen while watching for the appearance of the opaque substance in the intestine. In non-obstructive cases, the contents begin to traverse the pylorus at once. When any doubt exists as to the patency, a film is taken four hours later. In complete obstruction no barium has passed the stomach. There can then be no doubt as to the diagnosis. In many cases a certain amount will be found in the intestine. The estimation of the amount of gastric retention is roughly made in terms of percentages. If at the end of four hours, as much as 50 to 75 per cent of the opaque meal is still in the stomach, a high grade of obstruction exists.

The child has a peculiar form of constipation. The stool contains little fecal material, is small in amount and consists mostly of mucus. There may be one or two such small movements or a day may elapse without any stool. It is the type found in protracted starvation.

The urine is likewise scanty because of deficient absorption of food and liquids. Acetone and diacetic acid may be present.

Another cardinal symptom is that of hyperperistalsis. During or soon after feeding or drinking, peristaltic waves will be visible. This gastric phenomenon is due to increased stimulation or spasm of the stomach wall. Tumor-like elevations pass across the upper abdominal region beginning at the cardiac portion of the stomach below the left costal arch and ending at the pyloric end near the right mammary line. The peristaltic wave occurs spontaneously, or it may be brought on by tapping the abdominal wall over the cardiac end of the stomach. The entire outline of the stomach may be at times observable, not only in peristaltic motions, but as a spastic contraction. These manifestations give a fullness to the region of the abdomen above the umbilicus which is in contrast to the flat or sunken lower abdominal area.

The pyloric tumor itself may be palpated if of sufficient thickness or hardness. This is favored not only by the thinness of the abdominal wall but by the proximity of the tumor to the surface. At operation the dilated stomach and the pyloric portion usually present themselves as soon as the peritoneum is divided. There are certain shapes of the tumor seen at opera-

tion which are truly hypertrophic but not markedly bulging. The typical olive-shaped, hard, circumscribed mass lends itself to palpation better than the less definitely thickened type. The physician is not always successful in palpating the tumor. Its situation is just to the right of the mid-abdomi-



FIG. 28.—RADIOGRAM IN PYLORIC STENOSIS SHOWING THE BARIUM STILL IN THE STOMACH AT THE END OF FOUR HOURS

nal line and above the umbilical level. The tumor grows larger as the disease progresses.

Loss of weight is a symptom as well as a prognostic feature. Complete obstruction at the pylorus means a rapid and marked emaciation. If the infant has lost one-third of the body weight, the case has been one of marked severity and the outlook is not favorable. Recovery, however,

has been reported in losses exceeding 33 per cent. It is usual for the child to lose 1 or 2 ounces daily. This becomes a serious matter in the course of a few weeks. In some cases there may be remissions in the pyloric symptoms with utilization of a portion of the food. The weight will then remain constant or temporarily show an increase.

Most cases have no elevation of temperature. The mouth may become dry and the lips red. Dehydration and air hunger are present in some instances.

So far as is known the infant at no time suffers from pain. The anxiety



FIG. 29.—CONTRACTIONS OF THE STOMACH SHOWING THE PERISTALTIC WAVES IN PYLORIC STENOSIS

is shown in the "drawn" facies. When the disease has lasted several weeks, the appearance of the infant is pitiable and miserable.

Tenacity to life is remarkable and recovery may take place even when operation is undertaken in extreme states. The writer recalls one instance where the emaciated infant had apparently stopped breathing, but with the consent of the mother the abdomen was opened without any anesthetic, the muscular fibers of the pylorus were incised, the external wound quickly sutured and the infant put back in a warm bed. Immediately the child's condition began to improve and an uneventful recovery took place. The following is a typical case with operation and no untoward incidents during convalescence:

M., four-weeks-old boy, entered the hospital with a history of vomiting all feedings at the breast since two weeks of age, with a marked loss of weight, with scanty or no daily stools. Inspection of the abdomen showed marked patterns of the stomach and hyperperistalsis especially noticeable immediately after feeding. The vomiting was projectile. Radiogram four hours following

barium in buttermilk revealed the retention of at least 95 per cent of the barium in the stomach, much of it still there at twenty-four hours, and only a trace in the intestines. The child was markedly dehydrated and toxic.

The only abnormalities in the blood were the high urea nitrogen, 28.7 Mg. (the normal being 12 to 20 Mg.) and the rather high  $\text{CO}_2$ , 73 per cent (indicative of alkalosis, the normal in infants averaging 50 per cent).

The urine was normal.

It was thought advisable to try the effect of atropin and thick cereal feeding for two days, but vomiting continued. Because of the lack of retention of all food and fluids, physiological salt solution was given under the skin and a pint or more was readily absorbed in this way. (This is a valuable procedure in cases of continued vomiting, especially as a preoperative measure.) In addition the infant was given 200 c.c. of suitable citrated blood in the vein at the elbow. The result of the transfusion was an increase in the blood content and an improvement in the color of the skin, and in the operative resistance.

The classical Fredet-Rammstedt operation was performed twenty-four hours after the transfusion, the anesthetic was novocain,  $\frac{1}{2}$  of 1 per cent locally.

Fluids were administered in teaspoonful and increasing amounts each hour after the operation, and at eight hours the baby was put to the breast. He nursed vigorously and took both breast and thick cereal feedings thereafter until the mother's milk was sufficient for his needs. His weight decreased for two days, then began to increase, and at one week he had gained five ounces. The subsequent course was that of a healthy, thriving infant.

The course of the disease is from four to six weeks, but with proper medical or surgical treatment it will naturally be shortened. Beginning convalescence is evidenced by a reduction in the vomiting, by improvement in the weight, in the size and character of the stools, and in the volume of the urine.

In unfavorable cases, intoxication, starvation or intercurrent infections such as pneumonia end the infant's life.

**Diagnosis and Differentiation.**—It is common for some time to elapse before the true nature of the disease is discovered. Dietary indiscretions of the mother and colic on the part of the infant may be blamed for the digestive upset. The physician who has previously followed a case from the beginning is not apt to overlook the importance of early symptoms. The clinical picture leaves little room for doubt as to the nature of the affection. The physician should not hesitate to pass a catheter into the stomach and determine the retention. This should be done four hours after feeding. In spite of the fact that four hours have elapsed since feeding and that vomiting has occurred, a large amount of residue will be found in the stomach. This may exceed the amount taken at the last meal. In doubtful cases repeated observations of this kind are useful.

Early in the disease there will be time to secure the assistance of the Roentgen ray in diagnosis. To the physician experienced in the clinical signs, this is not absolutely necessary except where doubt exists. When the pylorus is patent, the opaque meal will begin at once to enter the duodenum. At the end of four hours most of the barium should be in the



intestines. An estimation of the percentage passing the pylorus is used by Strauss to decide the question of operative interference. If less than 30 per cent has passed in four hours after feeding, operation is indicated.

Other causes for vomiting at this early time of life must be excluded. Simple vomiting in the nature of regurgitation from too frequent or too large feedings is entirely different in type from projectile expulsion of food. It is corrected by lengthening the intervals between nursings and by reducing the number of minutes at the breast. The colicky or hypertonic infant may have irregular vomiting, not occurring so promptly after nursing and not accompanied by a steady loss of weight. In fact such an infant may at times gain sudden large amounts and may be well nourished. Such an infant has intolerant crying and has a history, both personal and family, of a neuropathic diathesis. So far as medical therapy is concerned, the so-called pylorospasm need not be differentiated from true hypertrophic stenosis. No tumor will be felt and the obstruction will be intermittent. It is amenable to sufficient dosage of atropin and to the feeding of thick cereals.

Toxic vomiting from infections within or outside of the intestinal tract has an absence of obstructive symptoms. Projectile vomiting is a symptom of brain tumor and especially of tuberculous meningitis, almost unknown in the early weeks of life. Here the vomiting is not constant and has no reference to meals. Constipation is present also, but the stools are not small and are not of the starvation type. Appendicitis is rare at this age, but its diagnostic signs should be borne in mind: an acute course, initial vomiting, pain, tenderness, abdominal distention and peritonitis, leukocytosis, fever and a mass felt low in the abdomen.

Low intestinal obstruction due to congenital atresia and other malformations shows an obstruction to the opaque meal at these lower levels. When duodenal atresia is present the barium may pass the pylorus, but will stop in the blind end of the intestine. Intussusception occurs at a somewhat later age than pyloric stenosis, and it is characterized by acute vomiting, bloody stools and a sausage-shaped tumor in the lower abdomen.

When persistent vomiting occurs in the infant soon after birth, especially in the first few days, one should bear in mind the possibility of a congenital obstruction elsewhere than in the pylorus. One of these is the above-mentioned malformation of the duodenum. Another cause of prompt vomiting is the deformity or atresia of the esophagus. The writer has seen three cases of this type. Beginning with the first feeding of water, the infant vomits all fluids. This should at once suggest obstruction in the esophagus and can be readily confirmed by the inability to pass a small-sized catheter (from a No. 12 to 16 French).

Above all, it is well to remember the age at which pyloric stenosis usually appears.

**Treatment.**—The question of medical versus surgical treatment is one in which opinion varies. Johannessen, of Denmark, is opposed to surgical treatment. He uses atrophin in large doses and states that the effect may not be attained until the tenth to the fourteenth day of treatment. Herz, of Germany, recently reported fifty-two cases, not operated upon, none of whom died from obstruction and half of whom were cured without any drugs. His optimistic report is not in accord with those observers who insist upon a pyloric tumor and persistent obstruction as the basis for treatment.

The British profession is generally in favor of immediate operation when the diagnosis is complete. When the mother has plenty of milk, Still insists that operation should be done at once so as to interrupt the breast feedings only on the day of operation. Ashby and Southam favor the surgical method of treatment in the hands of an experienced operator. They object to the long convalescence during medical treatment and the failure in many cases, as contrasted with the rapid cure, marked nutritional gains and disappearance of the tumor following operation.

There has been marked improvement in all forms of treatment and the conservative opinion in America is that the question of the type of therapy should be based upon the amount of obstruction, the welfare of the child and the urgency of prompt relief. In the hands of experienced clinicians, the medical or dietary treatment has usually given good results, but most observers have recognized the need for surgical operation in definite cases. The deciding factor is largely a personal one both as concerns the infant and the experience of the physician or surgeon.

Treatment begun in the early days of the vomiting permits of much difference from that employed later. When an infant is brought with a portion of the food passing the pylorus, the nutrition still good and little evidence of toxemia, it is safe to try the effect of nonsurgical methods.

**Stomach Washing.**—Gastric lavage with warm water once or twice daily an hour before feeding is of some value. It not only empties the stomach of residual debris, but renders easier the passage of food when the obstruction is not complete. It is especially indicated early in the course and should be persisted in as long as any benefit is caused thereby.

**Dietary.**—At first the interval between nursings should be lengthened to four hours. The time at the breast is reduced to five minutes or less. If no reduction in vomiting occurs, thick cereal cooked with water for one hour,  $\frac{1}{2}$  to 1 ounce, is fed by a spoon immediately after a short nursing of a few minutes. Another method is to alternate the breast nursing with a gruel feeding. The breast milk may be expressed and stirred into the thickened gruel. Skim milk may be cooked with the cereal if breast milk is unavailable.

Thick feeding should be begun in every case, as it lowers the tendency to vomit, whether the treatment be surgical or medical. Epstein and Rogatz

state that thick cereal should not be made up with milk because of the liquefaction of the mixture in the stomach. Cereal alone is obviously unsuited for long-continued administration, and should be alternated with some milk-containing nourishment.

The infants tolerate easily the well-cooked starchy foods. Grulee has warned against the danger of water starvation of infants fed solely upon concentrated foods. Water may be given two hours after the thick food with the expectation that some of it will be utilized. It is well to administer daily by hypodermic injection a pint of sterile water or Ringer's solution.

Recently there has been published in the German literature the record of three infants dying from asphyxia resulting from the respiratory aspiration of vomited thick gruel. At autopsy cereal was found in the fine bronchioles. Aspiration of concentrated food has not been reported in this country so far as the writer is aware.

Much attention should be paid to the technic of thick feeding. Sauer has written exhaustively upon this subject, and to him belongs the credit for much of the improvement in the dietary methods. His directions for preparing and administering cereal concentrations are briefly as follows:

Farina or rice flour .....	6 teaspoonfuls
Skim milk .....	9 ounces
Water .....	12 ounces
Dextrimaltose .....	3 ounces

Cook for one hour in a double boiler until the food is quite thick or pasty. Feed with a spoon or wooden tongue blade. Give from 2 to 4 tablespoonfuls every three hours during the day and twice during the night.

**Medical Treatment.**—Numerous drugs have been tried, but atropin is commonly used. The effect of this agent is the reduction of the general hypertonicity and of the spasm of the unstriated muscle fibers. A series of cases treated in this way has been reported by Haas. A convenient method is the use of a 1 : 1000 solution of fresh atropin sulphate beginning with one drop before feeding, and increasing the number of drops until the effect is obtained. Many infants develop erythema of the skin from atropin administration. Advocates of this drug emphasize the harmlessness of large doses, and persistence in its administration until the physiological effect is reached. Hypodermic injection of atropin has been found to give more regular results. Its use up to 1 : 250 of a grain by this method would seem to be a dosage beyond which one should proceed cautiously.

Atropin therapy has a limited field in my experience with hypertrophic pyloric stenosis. It should be given a fair trial along with dietary treatment in the mild cases as long as there is improvement.

Mention should be made of the advantages from the services of a com-



petent nurse experienced in the feeding and observation of the vomiting infant. Other factors may influence the retention of food. Herz advises the administration of food through a nasal tube as causing the least disturbance. This requires a nurse trained in such a technic. The posture of the infant may be changed to advantage, some infants retaining food better when lying on the stomach.

**Surgical Treatment.**—It will be found much safer, shorter, and the mortality rate much lower to have the infant operated upon at the proper time rather than to persist in the use of non-surgical methods. It seems logical and by experience preferable to seek relief and cure by operation upon the tumor.

*Preparation of the Infant for Operation.*—When the child is in good condition there is no contra-indication to immediate operation. When the risk is poor because of dehydration and emaciation, preoperative measures are advisable for one or two days. These are to be obtained in the hospital. Hypodermoclysis of 200 to 300 c.c. of physiological salt solution should be given. When it is possible to secure compatible blood from a donor, an intravenous transfusion of citrated blood will increase the operative endurance as well as supply immediate nourishment and blood volume. The amount to be given intravenously is 30 c.c. (1 ounce) for each kilogram ( $2\frac{1}{4}$  pounds) of the infant's weight. Although the veins are small they are usually seen in the emaciated infant. A small needle is required. Transfusion of young infants requires dexterity and experience.

The maintenance of body heat before, during and after operation also helps to prevent the loss of vitality and weight. While on the operating table the child should be protected by hot-water bottles and a warm room. Little blood should be lost and no bleeding vessels left untied.

Following the suggestion of Langley Porter no recent cases of mine have been given a general anesthetic. Local anesthesia requires a little more time and dexterity during the operation, but is always to be used because of its freedom from danger.

*Choice of Operation.*—During the early days of surgical treatment for pyloric tumor, gastrojejunostomy was the method employed. The operative mortality was so great that the infant's life was seldom saved. The Rammstedt modification of the Dufour-Fredet or Weber operation has become almost universally employed, because of its simplicity and good results. Strauss, of Chicago, has devised a successful plastic operation upon the pyloric muscle. It is equally efficient but requires much experience and particular skill, and in most hands a longer time. It has the advantage of completeness, and obviates the necessity for repetition.

No surgical procedure in early infancy has saved so many lives as the simple pyloric incision used in the Fredet-Rammstedt type of operation.

The Fredet-Rammstedt operation consists in a right rectus incision above the umbilical level, the delivery of the pylorus through the peritoneal



incision and a division of the hypertrophied muscular wall by an incision along the longitudinal axis of the pylorus. As soon as the muscular coat is opened, the mucous membrane will be seen to protrude through the gap. It is well to observe whether gastric contents begin at once to pass through the canal. The pyloric wound is then covered with omentum, after which the peritoneum, abdominal wall and skin incisions are closed.

*Postoperative Care.*—The vigorous infant may be put to the breast for a few minutes within an hour after the operation. With the toxic and weak child feeding should be begun more slowly. At the end of the first hour a few teaspoons of water are given, at two hours a like amount of breast milk, and thereafter each hour for the first day water and breast milk are alternated. Most infants have no vomiting after the operation. When this annoying and somewhat embarrassing symptom recurs it is well to continue the use of atropin and thick cereal until gastric retention is normal. Hypodermoclysis of salt solution may be needed in the severe case for a few days. The body heat should be maintained by hot-water bottles. In one case the persistence of vomiting necessitated opening of the wound and the extension of the pyloric incision. This freed the remaining muscular fibers which had not been sufficiently divided at the first operation. No further vomiting occurred. In emaciated infants breast milk is of great importance in the after feeding. In case the mother has little or none, a wet-nurse should be secured for a few weeks. In this way the infant gains rapidly. Artificial feeding may consist of cereal to which skim milk and dextrimaltose are added, with the gradual addition of a portion of the cream.

**Prognosis.**—Early treatment which checks the vomiting gives much better outlook and shorter duration whether the method is medicinal, dietary or surgical. The disease should receive prompt and persistent attention. The maintenance of breast feeding is highly desirable. A minimum loss of body weight is obtained. In 163 cases analyzed by Goldbloom and Spence the outcome was less favorable when the weight loss exceeded 20 per cent. Fortunately one sees numerous cases recover after much greater loss than this. Morrison reported recovery in an infant losing 50 per cent of its weight. A premature baby weighing  $3\frac{3}{4}$  pounds developed symptoms at thirteen days and was operated upon at thirty-five days, recovering rapidly thereafter.

It is impossible to give accurate mortality figures, as these vary greatly. There are deaths during non-surgical treatment which might have been prevented by early operation. There are also deaths hastened by late operation and others the result of surgical disasters and postoperative complications. Moore reports death from respiratory failure in eight days following operation in twins who weighed less than 5 pounds.

The statistics of Bolling deal with 454 cases operated upon by the Fredet-Rammstedt method with a death-rate of 15 per cent. The causes

of death are chiefly collapse and less commonly hemorrhage, peritonitis, acute gastro-enteritis, bronchopneumonia, accidental laceration of the duodenum and rarely intussusception. It seems reasonable that the mortality statistics should be based upon the operative risks, as early diagnosis and operation mean a far better nutritional state of the child, more successful results and fewer complications.

The death-rate is high in untreated cases. Difference in the severity of the disease depends upon the degree of obstruction and the rapidity of the course.

In favorable cases the recovery is ultimately complete. The writer has never seen a poorly nourished or abnormal infant at the age of one year after operation. The gastric functions in later years are found to be normal whatever the method of successful treatment.

The best results will be obtained by early operation. This is especially true when the physician has had limited opportunity for feeding such cases, and when he can secure the services of an experienced surgeon and a well-equipped hospital.

**Pyloric Stenosis in Older Children.**—True pyloric hypertrophy is rare in later infancy and older childhood. Jampolis showed a case in a five-year-old girl cured by gastro-enterostomy. The following is the history of the writer's case occurring in a nine-year-old girl:

A girl, aged nine, normal weight, 11 inches underheight, apparently of normal intelligence, but showing definite symptoms of hypothyroidism (dry skin, much coarse hair, lack of perspiration, phlegmatic temperament, lordosis). She had been getting thyroid extract since the age of eight months. Her symptoms had been vomiting of mucus each morning before breakfast, but the vomitus free of food elements. Examination with an opaque meal showed by the fluoroscope a markedly dilated stomach, vigorous peristalsis, and slow emptying time. At five hours 50 per cent of the residue was still in the stomach. At operation a true pyloric tumor was found. A gastro-enterostomy was per-



FIG. 30.—NINE-YEAR-OLD GIRL WITH PYLORIC HYPERTROPHY

formed and the child has since been free from vomiting and the emptying time of the stomach has been normal. The features of interest were the occurrence of a true pyloric tumor in a child of this age, with infantile development due to hypothyroidism.

## GASTRO-ENTEROSPASM OF INFANCY

(*Pylorospasm*)

**Clinical Description.**—The syndrome begins in the infant from one to two months of age, either breast or artificially fed, and may last for as many as six months. The two symptoms which are outstanding are the definite tendency to vomit and the marked restlessness and immoderate crying of the infant after feeding and until late in the night. Vomiting is not usually so forcible as in true pyloric stenosis and does not occur so regularly. The pylorus cannot be felt through the abdominal wall and no true tumor exists. Moderate peristaltic waves affecting the stomach and intestines may be seen in the thin-muscled infant. Alternate constipation and diarrhea occur. The nutrition usually suffers only slightly, when there will be periods of stationary or slight loss of weight. Vomiting may be severe enough to cause emaciation. The outcome is good and the treatment successful.

**Nomenclature and History.**—Until recently much confusion has existed regarding the identity of pylorospasm. Cases occurring in early infancy have not always been separated from hypertrophic pyloric stenosis, until coming to operation when no tumor or obstruction could be found. In my earlier practice, I have had two such experiences. It is to be hoped that in the future the term suggested by White, infantile gastro-enterospasm, will be accepted and used to supplant the confusing term "pylorospasm." "Vagotonia" has also been employed to designate the same clinical syndrome. In the eighth edition of Holt and Howland this tendency to tonic muscular spasm is described under the heading of the neuropathic infant. Haas and others have called it the hypertonic diathesis.

No extensive literature exists in which pylorospasm is adequately treated from its etiological and pathological characteristics. Much of a controversial nature, however, has been printed in which some writers doubt the existence of such a clinical entity, other writers believing that it is an earlier stage of true pyloric stenosis; and others believe that any case treated successfully by medical or dietary measures proves the absence of hypertrophic stenosis and the presence of an entirely different disease.

**Etiology.**—It is improbable that spasm would affect the pylorus alone. There is at operation no malformation of the pylorus, and the disease is not therefore one for surgical intervention. The entire gastro-intestinal tract is involved in the increase of nerve tone, and producing likewise general hyperirritability. The theory as supported by White is that there is a lack



of balance between the stimulating action of the vagus system and the inhibiting action of the sympathetic nervous system. Reyher is of the opinion that spasm of intermittent character does occur and affects the esophagus, stomach and intestine in children who have increased vagus tonus or increased excitability of the vegetative nervous system. The underlying cause is not understood. A familial tendency as regards other children or the parents is frequently found in the history. I have however at present under observation Negro twins, young infants, one of whom is quiet, fat and never vomits, the other cries much of the time, vomits, has spastic retraction of the head and spine, fails to gain, but has remissions at times, and becomes normal during the administration of atropin. Both infants are fed similarly at the breast. I have in certain instances discontinued the breast feedings to find no cessation of symptoms on other foods which are ordinarily well tolerated. The infants gain in weight but seldom become comfortable and quiet by dietary means alone.

**Physiology and Pathology.**—The vomiting is due to the gastric spasm. It is probable that the stomach and duodenum become somewhat dilated. Evidences of tissue changes are wanting. The entire gastro-intestinal musculature is involved in the spasm. Observation during operation furnishes the only information available, as these infants do not die or come to autopsy. Only in severe cases do intoxication, dehydration or wasting occur.

**Symptoms.**—Every physician is familiar with the picture of the infant who is abnormally wakeful, and who cries from late in the afternoon till well towards midnight. Among the laity it is known as three-months' colic. There is at times evidence of abdominal distention and undue amount of gas present. The infant may cry during nursing or thereafter, and seem definitely uncomfortable.

There are visible gastric and intestinal patterns due to hyperperistalsis, but no true peristaltic wave of the stomach alone.

The vomiting may appear at the same age as that of pyloric stenosis, but not so soon after feeding. It may be a simple regurgitation or of the mild projectile type. There are periods of remission when there is no vomiting. The emptying time of the stomach may be intermittently delayed, but there is no constant pyloric obstruction. When the barium meal is given it may rapidly pass the pylorus.

The stools may be undigested and loose, normal or constipated. They vary in the same infant.

The nutrition is not markedly disturbed. Usually the weight remains stationary for weeks, or during remission the gain is normal.

Certain cases have marked general tonic spasm, with characteristic backward bending of the head and flexion of the extremities. These infants are usually precocious and react unduly to noises and fright.

**Treatment.**—The infant should be kept undisturbed as much as possible, except for the regular feedings which should be reduced to five in twenty-



four hours. The vomiting, constipation, crying and sleeplessness are sometimes benefited by replacing one or two of the breast feedings with buttermilk. The buttermilk is to be mixed with an equal amount of thick cereal and fed with a spoon. A few spoonfuls may also be given after each nursing.

Considerable personal experience has convinced me that the use of atropin in sufficient doses will relieve all cases of true gastro-enterospasm, both as regards the vomiting and the general hypertonicity. Small doses may be of no benefit. There is much unwarranted timidity on the part of physicians, nurses and the family in the administration of this drug. Not infrequently some infants show flushing of the skin with small doses, but this is not injurious or dangerous. If a 1:1500 or 1:1000 solution is used the dose may be carefully increased so that one need not fear serious idiosyncrasy. Hamburger reported a five-month-old infant, weighing 7 pounds, that tolerated 1.8 milligram ( $\frac{1}{36}$  grain) without any effect upon the pupils.

A useful and careful technic in administering atropin is as follows:

R Atropin sulphate (fresh).....grain  $\frac{1}{4}$  (0.0162)  
 Distilled water ..... 5 ss. (15 c.c.)  
 M. Dispense in dropper bottle.  
 Sig. One or more drops as directed.

The dosage is begun with one drop before feedings. If no benefit is obtained on the first day, two drops at a dose are given on the second day, and increased daily until the effect is secured, it often being necessary to reach five, six or seven drops. An important point to remember is that continued administration is necessary to keep the child comfortable and free of vomiting. After a few weeks of relief the use of the drug may be left off, to be again begun if symptoms return.

Promiscuous handling by excited members of the household is to be avoided, as this reacts upon the infant. The mother has usually become tired and nervous and should be relieved of the care of the child. The assistance of a nurse is helpful. She should be of a quiet and not easily disturbed temperament. Much benefit will result if the child be taken outdoors for air, sunshine and sleep.

**Diagnosis and Differentiation.**—Irregular vomiting in the young infant, not accompanied by marked continuous weight loss, with symptoms beginning while on the breast, and with an abnormal amount of crying, restlessness and difficult sleep, make up the familiar picture. General nervous irritability and hypertonus are marked features in certain cases. There may be either constipation or diarrhea.

Hypertrophic pyloric stenosis has an abnormal gastric retention, almost complete pyloric obstruction, and a pyloric tumor. Peristalsis is more active. Nutritional loss, stupor, prostration, toxemia and constipation always develop.

The pronounced restlessness, crying and loss of sleep in congenital syphilis (Sisto's sign) occur in early infancy. It may be accompanied by vomiting and loss of weight. General hypertonus and gastro-enterospasm do not belong to this disease.

Vomiting from overfeeding is relieved by reduction in the size and frequency of the nursings. When the child is being fed unsuitable artificial food, there will be evidences of intestinal indigestion in the stools, which are hard or may become loose.

In the beginning the colic of hypertonic infants might be mistaken for the pain of appendicitis, but the latter results from an acute obstructive and inflammatory process with fever and leukocytosis and should be borne in mind when crying and vomiting occur in any child.

**Prognosis.**—Because of the slight amount of disturbance in the nutrition and of the absence of anatomical change in these children there is usually no impairment of the later health. Some of the symptoms last until six or nine months of age. No child dies of this affection. Hirsch thinks that atonic insufficiency of the stomach may be an after effect. Gastric dilatation and intestinal indigestion have been reported as sequelæ of pylorospasm. I have seen perfectly normal children grow out of hypertonus in infancy with no trace of hyperexcitability. Other children, however, may retain certain hypertonic characteristics, which are found in the neuropathic diathesis of later childhood.

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## CHAPTER XI

### RUMINATION AND CYCLIC VOMITING

#### RUMINATION

**Descriptive Summary.**—Rumination is a pernicious habit of food regurgitation and reswallowing, occurring in infants, less often in older children and occasionally in adults. The condition is apt to be overlooked in young infants as it may be regarded as simple spitting up of small portions of food. A small quantity of food is brought up, held in the mouth and pharynx where it is moved about with a gargling sound, and after a few seconds reswallowed. This cycle is repeated at frequent intervals. The habit may become persistent and be of serious significance as regards the nutritional state of the child, or may be an indication of mental impairment.

**Nomenclature.**—The symptom-complex is designated as rumination (from the Latin, *rumen* or throat) because of the resemblance of the phenomenon to that occurring in ruminant or cud-chewing animals. A synonymous but less used term is merycism.

**Etiology.**—Certain cases seem to be normal individuals at least at the onset. In others there is an underlying neuropathic diathesis. The practice is always a morbid phenomenon, and in older children or adults certainly an indication of some nervous or mental disorder. It is not uncommon in idiocy. Neuroses among members of the family are usually present. In certain individuals the only abnormal finding may be the evident desire for the pleasurable gratification that the practice affords. Strauch reports an infant of fifteen months who would regurgitate orange juice in order to taste it again, but who eventually extended the habit to other kinds of food.

The habit may follow the repeated occurrence of simple regurgitation, or the vomiting from diseased processes, especially pylorospasm or gastrointestinal disturbances. The practice may begin in children after infancy by imitation of the habit in other members of the family. Abnormal air-swallowing may be the cause of the early regurgitation. Air continues to be swallowed throughout the course, and increases the facility with which food may be brought up.

**Mechanism.**—The attacks may be involuntary or voluntary. In the beginning the regurgitation is involuntary. In some cases it has been noticed to happen while the infant is asleep. The efforts are often voluntary and are practiced for the satisfaction and pleasure of the child. There are two common opinions as to the underlying basis. First that there is no anatomic cause for the phenomenon, but that the symptom-complex is a neurosis.

The cure which results sometimes by psychic treatment would support this view. The secondary theory is that there exists a hyperirritability of the involuntary muscles of the stomach and esophagus. In certain cases the early history of the child indicates a spasm of the pylorus with forcing of food into the esophagus. The same tendency to spasm affects the circular muscles of the esophagus in which food accumulates to be afterwards regurgitated. If there is any pathologico-anatomic basis, it is probably in a secondary dilatation of the lower end of the esophagus. Bischoff described three types of rumination in infancy with all of which the esophagus is dilated near the stomach. This is shown by radiogram.

**Symptoms.**—*In Infancy.*—The occurrence is probably more common than the rather infrequent mention in the literature in this country would indicate. A certain amount of rumination is common in infants between three and six months. Mild cases are especially overlooked, as the rumination may happen only a few times in the course of a day, and the wastage of food is slight. There is no nausea or force connected with the bringing up of the food. Sucking of the fingers may be used to assist in the act.

Regurgitation is the first stage of the practice. Food is brought up in small amounts. When the habit is established and the typical symptom-complex observed, the infant is seen to suddenly become red in the face, with the respiration irregular, the head drawn back, the chest protruded, and the abdomen retracted. The mouth is opened wide and the tongue pressed against the lower jaw. This mechanism brings up a portion of the food, some of which escapes from the mouth.

The food is retained in the throat for a quarter to half a minute where it is moved back and forth into the mouth by the action of the tongue. A gargling sound is heard. Inspection of the pharynx shows the oscillation of the food to be rhythmical. If the child is put under the fluoroscope the up and down movement may be seen in the esophagus. These oscillatory movements average one each second, with occasional pauses.

In pronounced cases the ruminating act occurs every minute or two for the first half hour after nursing, and less often during the remainder of the interval between feedings. It may begin before nursing is completed. It is most easily accomplished when the stomach is full of food, water and air.

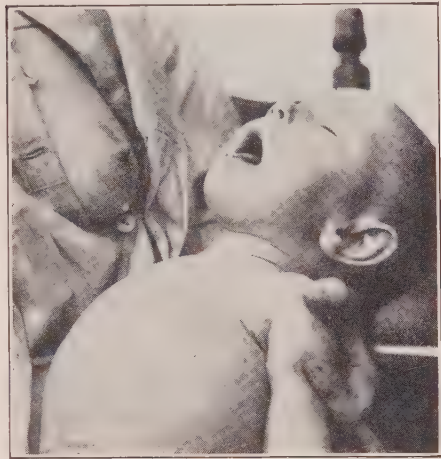


FIG. 31.—APPEARANCE OF INFANT DURING ACT OF RUMINATION

(Courtesy of Batchelor and Batchelor and *Am. J. Dis. Child.*, 1919, 17:43.)



Cases have been reported in which the child maintains normal weight and development. In such infants little food is wasted.

The nutrition is below normal in all cases that have been accompanied by spilling of food from the mouth for any length of time. The child is fretful and easily disturbed, and cries immoderately, and this further interferes with the weight. As would be expected when there is poor muscular and fat development, the temperature is below normal. The appetite is ravenous, and the child presents a pitiable appearance due to pallor and starvation.

**Course.**—Rumination often begins in the early months of infancy. The mild cases may recover promptly or gradually, or may pass into a severe type which is exhausting and intractable. Many of these infants, probably one-fourth of them, will die.

**Diagnosis.**—One should suspect rumination in the neuropathic infant who is undernourished. Grulee has called attention to the similarity in the general appearance of severe cases to that of marantic infants.

**Treatment.**—*Psychic Methods.*—The symptom-complex may be arrested by providing a new stimulus which attracts the attention of the infant. Psychotherapy is often used, first suggested by Schippers. The child should have new interests to take the place of the old. A change in environment will in some cases be of benefit. The child is given something to play with, or other occupation to attract the attention. Keeping the child out of doors for part of the day, especially soon after feeding, will cause fewer attacks. The child may at other times be isolated, where it may be distracted by new sights or sounds. A change in attendants may help for a while, especially the employment of a nurse. In older infants discipline is important. Some cases do better when the position is varied, the child lying part of the time on the stomach, or held in the arms while walking around—this is simply a method of bringing the child's attention to something new.

For the same reason other means of distracting the child may be valuable. A change in food to something thick, such as cereal, a hard cracker or piece of zwieback, is often useful. A capsule containing a grain of quinin may be given while feeding. The bitter taste on regurgitation may discourage further attempts at the time. Removal of tonsils and adenoids makes swallowing painful and difficult for several days. Circumcision also provides a new external stimulus. These are of only temporary benefit but may be enough when followed by other efforts as above suggested to cause a gradual or sudden cessation of the habit.

*Mechanical Methods.*—Keeping the fingers out of the mouth is important. The prevention of air-swallowing is difficult, but it may be facilitated by feeding the child in the upright position, and on the abdomen afterwards, as air is less easily swallowed. Tube feeding through the nose will allow little air to pass. Thick foods have been recommended, as there is less air swallowed and it is more easily expelled without bringing up food.

Strauch reported a cure by plugging the nares of a five-month-old child with cotton. This is to be recommended when the infant's food is liquid. It causes discomfort and interference with breathing and acts in a psychic and mechanical way to prevent the regurgitation.

A canvas cap with tapes fastened tightly under the chin so as to keep the mouth closed for two hours after meals was suggested by Batchelor. Its use should be continued until the habit is permanently broken.

A rather heroic but successful method is the introduction of a catheter over the end of which a balloon or fish bladder is surely fastened. This is passed into the stomach after feeding, and inflated *in situ*. It is then drawn up covering the cardiac opening. Siegert has tried this procedure in several instances. In one infant who was emaciated and threatened with starvation, a cure was effected within two months.



FIG. 32.—CANVAS CAP USED FOR RUMINATION

**Drugs.**—Loss of sleep and restlessness may be advantageously treated by the use of small doses of sodium bromid. Local application of cocain solution in the throat is not to be advised because of the dangers from the drug. Bismuth has been tried with indifferent results. Cod-liver oil and iron are needed when anemia and malnutrition are present.

**Diet.**—Concentrated, thick foods containing a maximum of nutritious substances are indicated. They are less easily regurgitated. Every portion of this which passes the pylorus adds to the nourishment of the child. Farina or oatmeal should be tried. To this may be added the curds from milk, or a milk powder. Cod-liver oil and concentrated orange juice are necessary. Emaciated infants should be given some breast milk if possible.

**In Older Children.**—Mentally normal older children seldom have the ruminating habit, but there may be a recurrence of a habit formed in infancy, or it may follow severe digestive disturbance. The parents as well as the child show a neuropathic tendency. Rumination at this age is not uncommon in idiocy.

The food regurgitated is of a thicker consistency than in infancy, and when brought up into the mouth is chewed and reswallowed. There is no disturbance in nutrition as none of the food escapes from the mouth. The habit may continue into adult life, as reported in the case of Strauch.

The persistence of the habit in an otherwise normal child is due to lack of instruction. Association with vigorous healthy children will probably cause the older child to stop the disgusting practice. Proper habits should be taught and bad ones corrected. Strict discipline is needed to bring about a

cure. Such children should become interested in outdoor games and athletics.

### CYCLIC VOMITING

**Descriptive Summary.**—Cyclic vomiting is a syndrome consisting of continuous hyperemesis for several days, followed by an interval of freedom for weeks or months, and by numerous repetitions of this cycle. There is an absence of demonstrable disease. The acute attack is self-limited, after which there is immediate recovery of the normal gastric function. It is probable that many different diseases are erroneously classed as cyclic vomiting. The affection disappears gradually as the child reaches maturity. Although the disturbance affects the gastro-intestinal tract, the underlying cause is a periodic derangement of the metabolism.

**Incidence.**—The syndrome is rare, although well known. Most cases begin between the ages of two and five years and may last throughout childhood. Cases have been reported among adults, but it is probable that such have an entirely different etiology.

**Etiology.**—Nothing is positively known about the underlying cause. The appearance of the onset in childhood and the usual cure when adult life is reached are indicative that age is a factor.

In a few instances I have found that a parent had been similarly affected when a child. A neuropathic constitution is usually closely connected with this condition. An unstable nervous balance is present in the child and may also be evident in the parent. The affection is more apt to be in an only child or in the youngest member of the family. Such cases are almost never seen in the routine of dispensary or hospital practice, but are more apt to appear among the well-to-do classes.

In recent years the cure of numerous cases by correction of ptosis of the abdominal viscera leads to the conclusion that physical abnormalities of this sort may play a part by interference with normal function. Fatigue is known to be easily produced in these children, and overexertion is often recorded just before the acute attack. The overtiring of the abdominal muscles permits an accentuation of the visceral sagging. The normal emptying of the stomach and intestine is delayed by the lowering of the muscular tone, and by the change in the contour of the gastro-intestinal lumen. Sagging and the resulting stasis in the stomach and duodenum are known to produce kinking of the pyloric wall or of the pyloric-duodenal junction. Intermittent obstruction may also be due to extrinsic causes such as compression, peritoneal bands, or traction by a short mesentery.

Appendiceal disease has been offered as an explanation. Comby is of the opinion that chronic appendicitis is a cause. This supposition removes the case from that of cyclic vomiting, which has no local symptoms referable to intestinal inflammation. It is probable that there is no such disease entity as chronic appendicitis in children.



Infected tonsils and adenoids may be an exciting cause. Sedgwick and others came to this conclusion after good results were obtained from the removal.

The metabolic changes which initiate the acute attack are not understood. Until recently it was thought that the processes could be explained upon the theory of acidosis. This conclusion was reached because of the presence of acetone in the urine, or its odor upon the breath. As these may not occur till late in the attack, their development is more apt to result from starvation than from the primary metabolic disturbance.

The most commonly accepted theory is that of disturbed fat metabolism. Assimilation is below normal. The stools are said to contain more than the normal amount of fat. These children have been thought to be intolerant of cow's milk fat.

It has been suggested but not yet proven that the metabolism of sugar is reduced. Greer found that a young child with severe vomiting responded to a subcutaneous injection of 3 per cent glucose solution by a doubling of the blood sugar content and by the appearance of sugar in the urine.

There are no known pathological changes connected with the causation of this syndrome.

**Symptoms.**—Attacks of protracted vomiting occurring at more or less regular periods characterize this syndrome. The duration of the acute stage is from two to five days. The intervals are usually once a month in the first year or two, but may be more frequent or not oftener than every two or three months. As the course progresses the periods become as infrequent as once or twice yearly.

The acute attack is preceded by a loss of appetite, a feeling of languor and apathy and finally nausea. Vomiting then begins and occurs with each attempt at drinking or eating, and in spite of the discontinuation of all feedings. Much mucus is present in the vomitus on the later days of the attack. The violent retching produces some streaks of blood which are seen in the mucus. There is complete anorexia until the vomiting process becomes less active. Thirst is pronounced.

The general appearance of the child soon indicates marked prostration. Exhaustion occurs from the stress of vomiting and the starvation. The eyes become sunken and the facies anxious. The tongue is coated and the mouth dry. The breath may reveal the sweetish odor of acetone, or may be extremely foul. There is no fever during the course.

Pain in the abdomen is unusual, but soreness of the muscles is complained of, due to the straining and retching. The abdominal wall loses its tone and becomes sunken.

The bowels are inactive. Constipation soon develops, and persists throughout the attack, due to the inhibition of peristalsis and the absence of food in the intestine.

The urine is diminished. The characteristic in its chemical finding is



the marked reaction for the acetone bodies. This is most common towards the end of the attack. The blood is said to contain measurable amounts of acetone.

The acute attack gradually subsides. It rarely lasts longer than five days, one week being the longest I have ever seen. Convalescence is rapid. Constipation may persist for a few days. The appetite promptly reappears, and the child soon seems little worse for the seizure.

The later history of these children is characterized by a failure to reach or maintain a normal body weight. The child barely has time to regain what has been lost before another attack begins. Such periods of starvation for a week, repeated every month or two, have a lasting effect upon the nutrition during childhood.

**Diagnosis.**—True cyclic vomiting is comparatively rare. Such a diagnosis is impossible during the first attack and cannot be made with certainty unless there is a history of repeated typical seizures.

All other causes for vomiting should be excluded. Probably the most common is *acute indigestion*, in the delicate or susceptible child. There is a history of eating unsuitable food, and the attack is usually accompanied by fever. It is only when constipation exists with vomiting and attacks are recurrent that cyclic vomiting comes into question. The writer has noticed that so-called "bilious" attacks in certain children, consisting chiefly of recurrent vomiting, are usually due to *acute tonsillitis*. All acute infections will be made known by their particular symptoms.

The most common mistake is to overlook *appendicitis*, especially in the atypical case which has little fever or in which the symptoms are with difficulty ascertained. The previous history will be extremely valuable. Appendicitis in children may be recurrent but not chronic. There is a type of appendiceal irritation due to infestation with pinworms that will produce vomiting and few other symptoms.

Hookworm infection of the duodenum has been reported in a large series of cases in all of which there was recurrent vomiting.

The beginning of catarrhal jaundice is usually accompanied by vomiting for a day or two before the icterus makes its appearance. I have not known true cyclic vomiting to develop jaundice.

Latent hereditary syphilis may have sudden and unexplained attacks of headache and recurrent vomiting. It will not be amiss to search for evidences of syphilis and to have a Wassermann test in all cases characterized by obscure periodical gastric upsets.

In every case of so-called cyclic vomiting the physician should search for the cause. Incomplete intestinal obstruction due to the traction of the mesentery or to pressure from peritoneal bands, may prove to be remedial by operation.

**Prognosis.**—Uncomplicated cyclic vomiting is never fatal. Griffith reported death in two cases that developed nephritis. Those cases of

vomiting which end in nephritis are probably of renal origin from the onset, or the kidney lesions may be secondary to an intercurrent infection such as tonsillitis, sinusitis, otitis media or respiratory disease. Rachford has suggested that these individuals sometimes become the victims of migraine in adult life.

The outlook is for the complete disappearance of the periodic vomiting after the age of puberty. The persistence of neuroses and undernutritional states will be found in certain individuals.

**Treatment.**—During the attack little can be done to overcome the vomiting. Lavage with Ringer's solution twice daily is often employed, and I always use it in the hope that some of the fluid at least may be retained or passed through the pylorus before vomiting recurs. Vomiting occurs whether fluids are drunk or not.

As regards laxatives, they are seldom retained, and their indication is not clear. However, I like the suggestion of Langley Porter that milk of magnesia be given in crushed ice at frequent intervals, not with the idea that we are treating an acidosis, but that the urine may be increased and alkalized. As for the ice, it is greatly appreciated by the child, and may result in some water being kept down.

Sleeplessness, restlessness and exhaustion are often extreme. Comfort may be produced by the administration of one or two hypodermic doses of codein or morphin during the twenty-four hours. No other medication is indicated. The services of a nurse are valuable.

The water equilibrium should be maintained. This is not possible by oral administration. The nurse may be successful in giving proctoclysis, but the restlessness and frequent vomiting interfere with the retention of the colon tube, which is annoying to these children. The hypodermic use of physiological salt solution is practical in the hands of the trained nurse. From 1 to 2 pints can be absorbed daily in this way. Sodium chlorid solution not only supplies the needed fluid, but it replaces the chlorid losses in the tissues and blood, and combats toxemia. When this method is unsuccessful and the need for water urgent, the solution may be given directly into the peritoneum once daily, in amount from 8 to 16 ounces (250 to 500 c.c.).

**Dietary.**—Milk and all other food should be at once interdicted. Crushed ice is given in an attempt to relieve the thirst. Whether any water is retained, its ingestion does no harm. As soon as the vomiting becomes lessened, orange or other fruit juice may be added to the ice.

The administration of insulin at the beginning of the attack is worthy of trial. The cases treated by Greer are not conclusive for its use in cyclic vomiting, but they suggest a method of solution for a clinical problem that has heretofore resisted all efforts. A buffer solution of glucose, 3 per cent, is given by the vein, or a 10 per cent solution by rectum. As soon as it

begins to be absorbed, a few units of insulin are injected. Greer reported relief from the vomiting within twenty-four hours.

*Treatment of the Underlying Condition.*—Attempts at preventing recurrences have been made by a restriction of the diet. Partial or complete withdrawal of milk and butter fat has been tried in most cases. It has been difficult to estimate the results, for they have not been marked. Other fats have been withdrawn, likewise the amount of carbohydrates. In my opinion the feeding of a well-balanced diet offers as much prospect of success as will be gained by making the feeding one-sided. Cereals, fruits in plentiful amounts, lean meats, coarse breads, green vegetables, and a pint of milk daily will make a satisfactory dietary.

For several years I followed the custom of giving sodium bicarbonate throughout the interval. It has to be given in large doses if the urine is to be alkalinized, but the continued use may result in an alkalosis, and is to be discouraged. The results are not satisfactory.

The most encouraging method of recent treatment is that of correcting the posture of the child who has visceral ptosis. The "lanky" child with thin musculature whose stomach sags below the umbilical level, and whose back shows a marked lumbar lordosis is a fit subject for such treatment. Sherman reports cures which he regards as spectacular. The results secured by Talbot by the application of an abdominal corset brace and other postural treatment warrant the trial in all appropriate cases. My own experience while limited has been likewise highly favorable. The function of the gastro-intestinal tract is improved, and the vomiting attacks are cured.

The removal of infection in the tonsils, adenoids, or sinuses is to be urged, as certain cases of periodic vomiting will be cured thereby.

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## CHAPTER XII

### THE INTESTINES

**Anatomy and Physiology.**—*Emptying Time.*—In normal full-term infants the food takes an average of fifteen hours to traverse the digestive tract. In premature infants, the time is somewhat less, being about twelve hours. During diarrhea in young children the food passes much more rapidly, usually within ten hours.

Breast milk leaves the stomach more rapidly than cow's milk and this shortens the time required to pass through the whole canal. I have seen a typical cow's milk stool sixteen hours after a newly born baby was fed for the first time.

*Length and Position.*—The determination of the *length* of the intestines as a whole as well as of its various portions, is of anatomic interest chiefly. One cannot say that a child of a given height and weight will have an intestinal tract of a definite length, and it varies much in children of the same age. In the full-term newly born infant the average distance from the stomach to the anus is 338.5 centimeters (133.27 inches) as found in the twenty-five measurements by Scammon. The newly born infant has one-half the length of intestine possessed by the adult. The intestine is approximately eight times the height of the infant while in the adult it is less than five times. The proportion of the small to the large intestines remains at the same ratio throughout life. The small intestine is from five to nine times the body length and doubles by the time of puberty.

Another way of estimating the linear measurement of the small intestine is based on the sitting height of the child. The small bowel has been found to be ten times the height of the body from the buttocks to the top of the head. From this measurement the intestinal area has been computed. Von Pirquet used the *surface area* of the intestines for determining the quantity of food needed by the individual. This method has not been used to any extent in this country.

Scammon finds in infants the lower end of the duodenum resting in the pyloric notch of the stomach, and the duodenal loop descending to a point near the right crest of the ileum. In later childhood the annular shape of the duodenum is more commonly found.

Infants have a great variability in the length of the large intestine, found by Robbin to be from one-half to one and one-half times the height of the body. It is therefore possible for the unusual length, especially if it occurs in the sigmoid region, to be responsible for functional disturbances.



The relative lengths and positions of the different portions of the colon in early life are: ascending colon, relatively shorter than in adults, and lying behind or just below the left lobe of the liver; the transverse colon is relatively longer and lies transversely under the liver and coming forward to the abdominal wall; the splenic flexure is adjacent to the right side of the spleen; the descending colon is comparatively short and lies against the left kidney; the sigmoid flexure is large and prominent at birth and during infancy, and occupies correspondingly more of the abdominal cavity than in later life. An unusually long large intestine, especially the sigmoid, may be a factor in producing constipation.

The rectum is comparatively long in early childhood. It is much straighter than in adults, but inclines somewhat from the vertical.

The *abdominal cavity* in the infant is much different in shape from that of the adult. The shallowness of the pelvis, the presence of a portion of the bladder above the brim and the space occupied by the large liver of the infant reduce the vertical diameter of the area left for the intestines, so that the transverse diameter of the mass of intestines is longer than the vertical. At birth only the rectum is found in the pelvis, but in later childhood some of the small intestine enters the pelvic cavity.

*Abnormalities.*—The advent of fluoroscopic and radiographic examination of the gastro-intestinal tract by the opaque meal and by rectal injection has given increased knowledge of the normal shape and function of the digestive tube. Peculiarities and malformations likewise are disclosed by this method. Among these are abnormal shapes and location of the stomach and intestines, and disturbances in the filling and emptying time.

*Peculiarities of the Intestinal Structures in Childhood.*—The mucous layer is frailer but has a relatively greater blood supply. The submucous and the muscular coats and the nerve tissue are incompletely developed in the first two years of life. The muscular layer is particularly thin.

The intestinal villi and the crypts or glands increase greatly in number during childhood. Lymphoid tissue shows a marked development in the first year of life, and the follicles, whether solitary or in Peyer's patches, are relatively more numerous.

*Physiology of the Duodenum.*—The duodenal contents in the healthy infant are less alkaline than formerly supposed. An examination of the hydrogen-ion concentration has been found to average 6.2  $P^H$ . When diarrhea is present the reaction in the duodenum is acid ( $P^H$  5). The activity of the gastric and pancreatic enzymes may be greatly reduced, similar to the decrease in urine, when fever, vomiting and dehydration occur. Hot weather has a similar effect. Diarrhea, especially, impairs the amylase and trypsin digestion. This accounts for the benefit from reduced feeding at such a time. Normal infants have a much lower bacterial content of the duodenum than do those with diarrhea.

The secretion found in the duodenum of infants has been found to con-

tain bile salts in a percentage concentration varying from 0.2 to 5 per cent.

**Intestinal Digestion.**—As has been stated, very little digestion takes place in the stomach. In the intestine the food comes in contact with the pancreatic juices. The protein is digested by the trypsin and changed into peptones. The peptones are acted upon by the intestinal secretion, namely the erepsin, whereby these protein substances are split into amino-acids to be absorbed from the intestinal wall.

The normal bacteria of the intestine are necessary for the digestion of food elements. The splitting or digestion of proteids is especially aided by various microörganisms, among them the *B. coli*. The intestinal and pancreatic ferments act upon starches and sugars converting them into glucose.

The splitting of fats occurs in the small intestine by the action of the bile and the pancreatic juice. As a result of the action of the digestants fat is changed into fatty acids and soap. Most absorption takes place through the wall of the small intestine, although it is a clinical observation that fluids containing salt and sugar may be taken up by the large intestine.

**Protein Digestion.**—Protein is digested by the gastric, pancreatic and intestinal juices, the end products being the amino-acid products which enter the circulation, and are taken to the tissues of the body. The splitting into amino-acids occurs in the intestine. These acids are necessary for growth and as fuel for energy. Certain proteins may not be completely broken down during digestive disturbances, and may enter the circulation in this undigested state. Those from animal sources cause better growth than from vegetables. That of mother's milk is superior to cow's milk and requires only one-half or one-third the quantity. Holt and Fales have emphasized the importance of providing in the food of children those proteins which have enough of the important amino-acids. Children need 44 grams ( $1\frac{1}{2}$  ounces) daily in the second year, increasing amounts each year until at the time of puberty, the large quantity of 130 grams is needed daily to supply maintenance and growth.

**Fat Digestion.**—Good digestion and absorption of large amounts of fat take place in the healthy infant. Little digestion occurs in the stomach by the action of the gastric lipase. Large amounts of fat may interfere with the gastric secretion and the emptying time. Emulsification and fat splitting take place in the small intestine. The cleavage of fat results in fatty acids and glycerin, which unite with the alkalies of the intestine to form soaps. The pancreatic secretion and its activator, the bile, are responsible for fat digestion. Large quantities of fat are found in the stools of normal infants, about 10 per cent being excreted. The percentage passed by the bowel decreases in older healthy children. The administration of high calcium food does not affect the absorption. The stools of infants who are fed cow's milk may contain fatty soaps due to incomplete digestion or to the combination of lime and fatty acids. Insoluble soaps thus formed

do not necessarily imply that there is a nutritional disturbance but in the presence of a stationary or decreasing body weight, it is advisable to reduce the amount of cow's milk fed to the child, and replace it by the addition of carbohydrates.

Fat is not well absorbed in certain diseases such as marasmus (athrepsia), tuberculosis of the intestines, celiac disease (chronic intestinal indigestion), and in diarrhea. In the latter, because of the increased peristalsis, fat is hurried through the intestines without time for absorption. Its utilization is not so high in cow's milk as in breast milk, so that the infant must take more cow's milk than breast milk in order to gain.

*Sugar Digestion.*—All carbohydrates are changed during the process of digestion into products which are absorbed chiefly in the small intestine, enter the circulation and become stored in the liver and muscles as glycogen. Intestinal bacteria act upon sugars to produce acids and alcohol. The inverting enzymes of the intestinal juices also convert sugar into absorbable forms. Diarrhea results from increased acid production, from deficient sugar digestion or absorption. Normal quantities in the food are handled without indigestion. A sufficient amount therefore is necessary to prevent bacterial action upon the protein and wastage thereof.

*Starch Digestion.*—From the first few days of life starch is digested and the tolerance steadily increases. Amylose, the starch-digesting enzyme, is present in the feces in slight traces, but increases with the age of the child and the use of starchy food. Starch digestion is begun by the ptyalin of the saliva some time after reaching the stomach, but more completely in the duodenum from the action of amylopsin. Since the advent of cereal feeding in vomiting infants, especially in pyloric stenosis, much has been learned about the digestibility of starch during early life. It has been found that well-cooked farinaceous foods, such as barley, farina and oatmeal, are taken without disturbances. Simchen tested with iodine all stools in infants for a period of four years, and while a residue of starch might be found in the stools, the incomplete digestion of a portion of it caused no disturbance.

Intestinal and other diseases may interfere with starch digestion. A definite symptom-complex resulting from overfeeding of starch has become known as starch injury (*Mehlnährschaden*). This is mentioned elsewhere in this volume.

**Stools in Infancy.**—*Newly Born.*—Meconium gives the characteristic green or black shiny appearance to the early stools. It is formed by the hepatic and intestinal secretions and the swallowed amniotic fluid. It therefore contains many elements, among them vernix caseosa, epithelial cells, fat and hair from the surface of the child's body, as well as bile, mucus and cells from the digestive tract. The examination of the meconium shows mineral salts present, among them iron, calcium, phosphorus and sulphur. Bacteria are absent at birth but appear in a few hours thereafter.

The total amount of meconium is usually from 2 to 3 ounces. Much



of it is voided on the first day, and it becomes replaced by fecal material within three to five days.

*Fecal Stools.*—Breast babies normally have bright yellow stools which are granular or pasty, and the odor is sour, aromatic and not offensive. To one who has observed stools the general type of the food can be told by the appearance and odor of the passages. When blue litmus paper is laid upon the breast-milk stool, the high acid content quickly permeates the paper and turns it red. Many breast babies have atypical stools, characterized by a greenish color, much water and mucus.

The normal yellow color is due to bile. If the stool is green as passed, the change has been caused by an oxidizing process within the intestinal tract whereby the bilirubin is changed to biliverdin. Diarrhea with increased peristalsis frequently gives rise to green stools. When the movement which at first is normal becomes green after standing, the bile salts therein have become oxidized.

In *artificially fed* infants the color is dependent upon the character of the food, especially the carbohydrate. Malt food is apt to make the stools brown or dark, while other sugars do not change the color. Grape juice, spinach, and drugs, particularly calomel, bismuth and iron, cause dark or black movements.

*Reaction.*—The reaction of the stool in the breast-fed infant is always acid, and except in overfeeding this never becomes harmful. The acidity is due to the presence of volatile acids, formic and acetic.

In the artificially fed infant the reaction varies with the food. Protein and skim milk usually cause neutral or alkaline stools, while food to which sufficient carbohydrate is added produces sour stools. The quantity of milk or of the additional sugar will affect the reaction. The practical significance of this fact is found in the dietary treatment of fermentation, diarrhea, constipation or putrefaction. Foods containing high protein and added sugar cause less acidity than low protein-sugar diets. Additional carbohydrate is less apt to be irritating and fermentative when fed with soured milk.

The presence of illness, such as parenteral infections (outside the intestinal tract), or digestive disturbances from intestinal injury, will bring on or aggravate excessive fermentation of the stools.

One can conclude that pasty stools are alkaline, as are also mucous stools in the absence of evident fermentation. At times fluid or soft stools are highly alkaline, but the presence of gaseous, sour and irritating stools indicates fermentative and acid changes.

*Fat.*—The gross appearance of the stools often indicates the presence of excessive fat. The movement may be oily and when spread on paper leaves a greasy stain. In the breast baby the small yellowish-white curds are fat. It has been estimated that there is an absorption of over 90 per cent of the fat taken by the breast baby, the total stool, however, when dried and



weighed being from one-third to one-half fat. High fat in the mother's milk gives the best stools in the normal infant. In the bottle baby the character of the fatty stool may be dry, crumbling, granular, or gray and mushy, with an offensive odor resembling cheese or butyric acid.

The amount of fat in the stools depends upon the diet. Milk-free diets will normally have little fat pass in the stool, but its presence indicates abnormal digestion or assimilation. The microscopical and chemical examination of the stools cannot be said to be of practical importance. The appearance of many fat globules in the stools of babies fed largely upon milk is of little significance as in infancy such a diet is normally productive of a certain amount of fat in the movements. In older children who are thriving normally and taking a mixed diet, little fat is left to go through. Therefore a large amount passed by children beyond the age of infancy has more significance. Acute or chronic indigestion causes a marked wastage. Holt found that watery stools decreased the amount of retained fat, and that the fat soap of the stool is replaced by fatty acids and neutral (unchanged) fat.

*Blood.*—The most common serious cause for hemorrhage in the stool is intestinal infection, while a not infrequent trivial cause is the stretching and tearing of the anal mucous membrane by a large hard fecal mass, which is streaked with blood. Hemorrhoids and prolapse cause a tinge of blood. Intestinal parasites and ulcers may cause gross or occult blood. Intussusception and Henoch's purpura cause a copious hemorrhagic stool.

*Salt Content.*—The amount of mineral salts, chlorid and nitrogen in the stools was found by Wang and Davis to be greater in the artificially fed child and during diarrhea. Urobilin is always present in the stools of normal infants and represents the amount of blood destruction going on in the body.

**Bacteria of the Infant Intestinal Tract.**—Much has been written about the influence of intestinal microorganisms in health and in disease. The gastric contents, the secretions and contents of the duodenum secured by catheterization, and the feces withdrawn from the sigmoid and rectum have furnished material on which examinations were made. Normal infants fed upon breast milk alone and others who were artificially nourished, as well as sick infants of both classes, have been studied. The results of some of the recent work and the practical conclusions therefrom will be briefly discussed.

Normal and probably pathogenic bacteria thrive in the intestinal canal, and have much significance. Bacteria are necessary for the digestion of the food elements. The normal stomach and duodenum contain relatively few bacteria, while the lower small bowel and the large intestine are rich in organisms. Both living and dead microorganisms are found in the stools. It is interesting to note that one-fourth of the stools when dried consists of dead bacteria. Nursing infants according to Brown and Bosworth ingest both staphylococci and *B. bifidus* (lactic acid organism) from the

mother's nipples, and these are found in the infant's stools. The micro-organisms are chiefly those which thrive best on the high sugar of breast milk.

In the artificially fed infant there is opportunity for a larger bacterial flora. Microorganisms are introduced from the outside, especially in the milk and water, while the chemical character of the food has a definite relation to the type which thrives best. Fermentative microorganisms grow best on the carbohydrates in the food, and they produce acids. Protein-splitting organisms thrive more abundantly in a food rich in protein, from which there results an alkaline and putrefactive intestinal content. The bacteria which are normally in the intestine will become acid-forming or putrefactive, depending on the character of the food. This is a characteristic of infancy.

Much speculation has existed as to the pathogenicity of the colon bacillus. In the artificially fed infant the Gram-negative types, such as the colon bacillus, are predominant in health. Colon bacilli are said to be inhibited in their growth by the lactic acid organisms of buttermilk. Castel believes that the colon bacilli and intestinal micrococci are relatively harmless, except in the presence of increased amounts of volatile fatty acids in the small intestine, when peristalsis will be hastened and diarrhea develop. The evidence is conclusive that colon bacillus infection of the small intestine occurs only as a sequel to injury of the intestinal wall. Specific infections by such organisms as the *B. typhosus*, *B. dysenteriae* or the organism of cholera definitely occur.

It is probably nearer correct to regard the prevailing types of organisms in abnormal stools as harmless growths which have developed in a favorable medium. Davison has clearly shown that putrefactive and fermentative stools may occur in normal children, and have nothing to do with the cause of acute intestinal indigestion which is a digestive disturbance, or with dysentery which is a febrile disease due to the action of dysentery bacilli upon the intestinal mucosa.

**Peptic Ulcers of the Duodenum.**—Ulcer of the duodenum is less rare in infancy than in later childhood, but is more common than the similar lesion in the stomach. The reason that it is considered so rare is probably because it is not looked for or considered as a possibility. About two hundred cases have now been reported. Until recent years only a few writers have mentioned the disease, and then from autopsy findings. Ulcer is most commonly mentioned in connection with so-called melena of the newly born and in marasmus of somewhat older infants. Although the lesions may be multiple they are not to be confused with the ulcerations of dysentery which occur lower in the intestinal canal. Holt and Helmholtz have especially called attention to the necessity of including duodenal ulcer as a possibility among the intestinal diseases in early childhood.

*Incidence.*—Eleven cases were found by Flesch and Entz in infants be-

tween six weeks and six months of age among 364 autopsies upon children. Compared with adults few cases are found in the series of operations for this cause. Only one child was included in six thousand cases of duodenal ulcer mentioned by Proctor. Michaelson reported 1.5 per cent of children in 911 cases of both gastric and duodenal ulcer. In analyzing the reports of sixty-five cases in infants, Palmer found only five infants over five months of age, and an average of three and one-half months.

The writer has seen one case which was not diagnosed but found at autopsy, but I have become convinced that one will not rarely encounter obscure disturbances that are due to ulceration. The greatest difficulty in diagnosis is the inability to follow a case seen in dispensary or hospital practice sufficiently long or intimately to secure an accurate history or record of progress, while the acute cases die without opportunity to verify the findings. I recall a breast-fed infant of one month of age, who had been vomiting many of the feedings for a week. Pyloric stenosis was excluded, and no hemorrhage occurred. The character of the food was then changed to a fat-free breast milk by expressing the milk and skimming it. The feeding of this was followed by a cessation in the vomiting. At the age of two years, the child died of an acute intestinal intoxication. At autopsy an old scar was found in the first portion of the duodenum which represented a long-healed ulcer. The association between the vomiting in early infancy and an ulcer of the duodenum seemed to be close if not positive.

The cause has been ascribed to infection with a diplococcus or streptococcus. Helmholz regards the occurrence as epidemic. Disturbance in nutrition seems to be an essential predisposing factor.

*Location and Character of Lesion.*—The ulcer is usually in the first portion of the duodenum, most often in the posterior wall and near the pylorus. The lesions may be multiple. They are never below the papilla marking the entrance of the common duct. The tendency for gastric ulcers to locate in or near the pylorus, and for duodenal ulcers to be relatively close to the pyloric junction speaks for their similar peptic origin. The ulcers vary in size from a pinhead to one-third of an inch in diameter. The ulcer may have a punched-out appearance, or may have sloping edges and be superficial. They do not have true indurations or marked round-cell infiltration. Kennedy states that ulcer is easily overlooked at autopsy in the fresh specimen, but when the duodenum is hardened in a fixing solution, the ulcer becomes evident.

In so-called melena of the newly born, the blood may come from this favorite location for duodenal ulcer. Until recently melena has been regarded as void of gross local lesions in the intestine, and as due to a hemorrhagic tendency. The finding of ulceration in certain cases shows that simple oozing may not always be the cause of this intestinal hemorrhage in infants.

*Symptoms.*—Duodenal ulcer is usually characterized by the appearance of blood, either manifest or occult, in the stool. Late in the case there may be blood in the vomitus. The child vomits persistently. This symptom is marked and is characteristic. It is probably due to the usual location of the ulcer just below the pylorus, and is often mistaken for pyloric stenosis.

Marked importance should be placed upon the peculiar cry of the infant which is of a persistent, wailing type, and is due to pain, which is further shown, coincidentally, by the drawn facies and the flexion of the legs.

The affected child is usually an infant that has been extremely restless, has pronounced anemia, and has been undergoing emaciation. The temperature is subnormal, and the fontanel depressed. The urine is reduced in amount.

Ulcers are usually acute, and the prognosis is poor when the nutrition is bad and hemorrhage is present. Infants do not stand any considerable loss of blood, death resulting rapidly within a few days.

Local or general peritonitis follows the perforation of the intestine.

A case reported by Thoms was in a five-month-old boy, artificially fed, small, atrophic, who had always had a tendency to vomit. The child suddenly developed bloody vomiting, hemorrhagic stools and died. The necropsy revealed a punched-out ulcer in the first part of the duodenum, near the pylorus. The ulcer was  $\frac{3}{8}$  inch in diameter, was irregular with undermined edges. The floor of the lesion consisted of submucous tissue and contained the bleeding vessel. There was no thickening, inflammatory reaction or peritonitis.

*Diagnosis.*—In the early weeks of life vomiting suggests pyloric stenosis. There is no tumor present in the region of the pylorus, and peristaltic waves are seldom noticeable.

Ulcer should be considered in any marantic infant if vomiting or hemorrhage persists. Hemorrhage is not evident in all cases, and its absence makes more difficult the recognition of this as yet neglected disease in infants.

*Treatment.*—Successful results in infants have been reported from the operation of gastrojejunostomy.

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## CHAPTER XIII

### THE INTESTINES (*Continued*)

#### DIARRHEA

**General Consideration.**—The tendency to diarrheal disturbances in early childhood is much greater than at any other period of life. This depends upon numerous factors. First of all it must be remembered that diarrhea is only a symptom of many diseases in which there is excessive motility and peristalsis of the intestines, as a result of which food is hurried through not entirely changed. The incomplete digestion of sugar and fats produces irritating fatty acids. The consistency of the stools becomes liquid due to the abnormally large secretion from the intestinal mucosa.

Food which is well borne in later childhood may cause diarrhea in infants, whose digestive tract is highly susceptible to injury by food or infection. The younger and frailer the infant the greater this tendency. Premature infants are particularly vulnerable, so much so that there may be an intolerance even for breast milk. The mucous and muscular coats of the intestines in the premature and congenitally weak infant are thin and delicate at birth. It has generally been considered that there is a certain amount of immature development of the intestinal tract in all newly born infants. Anatomically this is true, but functionally the full-term infant handles the kind and amount of food needed for its time of life. It has always been relatively easy to find a complementary food that is well tolerated by the infant who receives some breast milk. Bachmann emphasizes the fact the intestinal tract is sufficiently mature to digest such additional food as is now commonly given to the newly born infant, while awaiting the appearance of breast milk. It cannot be too strongly urged that this practice of early artificial feeding must be carefully controlled, so that as soon as possible complete nursing at the breast may not be embarrassed.

Of great importance in the prevention of diarrheal diseases in early life is the feeding of the infant at the breast. It is the common experience that no diarrhea of any seriousness ever develops in the full-term infant who is fed exclusively on the breast in proper amounts. Overfeeding will cause diarrheal stools in some infants, chiefly in those of the neuropathic (hyper-tonic) diathesis, due to an underlying irritability of the nervous mechanism of the gastro-intestinal tract and the tendency to hyperperistalsis.

The infant mortality statistics of the United States show a large decline in the number of deaths from diarrhea and enteritis. In the registration

area for the year 1915 there were 21.9 deaths from this cause for each 1,000 births as compared with 12.2 deaths in 1923. Each year the table of figures shows a constant decrease. The following table gives the death-rate from diarrhea and enteritis of infants under one year in the United States registration area for six years:

<i>Year</i>	<i>Death-Rate per 1,000 Births</i>
1918 .....	19.0
1919 .....	15.7
1920 .....	14.9
1921 .....	13.5
1922 .....	11.7
1923 .....	11.5

Minnesota has obtained an enviable reputation for infant welfare, since the public has been educated in the advisability for maternal nursing of all infants. The propaganda begun by Sedgwick, and other pediatricians, has been carried on by the State Board of Health. The result has been that infant mortality there has been reduced to one of the lowest in the United States. An organized effort is made to get every mother to nurse her infant entirely if possible.

DEATH-RATE FROM DIARRHEA AND INTESTINAL DISEASES, INFANTS UNDER ONE YEAR,  
CITY OF MINNEAPOLIS

Year	Number of Deaths	Rate per 1,000 Live Births	Rate per 100,000 Population
1909 .....	111	19.4	37.7
1910 .....	215	35.9	71.4
1911 .....	131	21.4	42.1
1912 .....	88	12.7	27.2
1913 .....	99	13.9	30.0
1914 .....	112	14.3	32.6
1915 .....	79	9.3	23.0
1916 .....	99	11.4	28.4
1917 .....	64	7.4	17.7
1918 .....	49	5.7	13.3
1919 .....	45	5.5	11.9
1920 .....	45	4.9	11.8
1921 .....	31	3.3	7.9
1922 .....	25	2.6	6.2
1923 .....	23	2.3	5.6
1924 .....	22	2.2	5.2

Although the percentage is steadily decreasing, it is capable of further improvement. Wynne, in England, found from the records of one hundred deaths in infants that twenty-four had been due to diarrheal diseases, thirty-nine from respiratory diseases, twenty-three from congenital weakness and abnormalities and fourteen from miscellaneous causes. In the slums he found that breast feeding is the rule among the poorest classes, and the

incidence of diarrhea is not so high in this class as in the next higher social class. There is a definite relationship between heredity, environment and infant mortality. The chief factor in environment is the feeding of the infant at the breast. Such feeding tends to overcome the handicaps of poverty, ignorance, poor hygiene and constitutional inferiority. The popular opinion that the children of the slums, some of whom survive in spite of filth, lead a charmed existence in the midst of an unfavorable environment is not borne out in fact. Breast feeding is their safeguard. Ignorance is usually an accompaniment of poverty and both increase the dangers from the accidents of feeding.

In the past the death-rate of artificially fed infants has been from three to five times that of breast infants, due especially to diarrheal diseases. Diarrhea is the disease of infancy which interferes most with a normal gain in weight.

Adequate reasons for failure to nurse at least partly at the breast are seldom found. Most premature weaning of the infant is done for trivial and illogical excuses, not alone due to ignorance on the part of the mother, but from suggestions and insistence by others. This advice may unfortunately be given by the physician, but is more often from friends or relatives. The necessity for gainful occupation on the part of the mother is a frequent reason for weaning. In any event the menace to the child is a real one when breast feeding in the early months is discontinued and indifferent artificial feeding substituted.

Artificial feeding, then, is responsible for much of the diarrhea in the infant. The breast-fed child will usually pass through an infection such as whooping-cough or pneumonia without intestinal disturbance; but in bottle-fed infants severe indigestion may appear. Overfeeding easily occurs from the bottle, for there is no limit to the amount which may be given in this way. I have found from experience that as the quantity of sweet milk approaches a quart in the twenty-four hours, and the addition of an amount of sugar which would be well tolerated with less milk, the formula is apt to be productive of gastro-intestinal disturbance. It will usually cause some discomfort, constipation, and subsequently undigested stools.

The influence of bad hygiene and ignorance must be considered as a cause of diarrheal diseases. The kitchen that is dirty and infested with flies, roaches and vermin, is not a safe place for the preparation and storage of infant food. Dirt in the bottles, upon the nipples, pacifiers and hands will carry infection.

Lack of knowledge in the care of babies is not confined to poverty, but it is less common among the well-to-do and is more capable of being remedied. In all classes there are mothers who are unfitted for handling children. Many infants are motherless, many are put in institutions, and all of these miss the advantage of proper home environment. It is a provision of nature that the infant may be fed at the breast of a mother possessing



little intelligence and who follows no rules in the feeding. The artificially fed baby needs the utmost care in preventing intestinal injury. The intestine of the healthy infant becomes more resistant with age but an attack of diarrhea makes the child more susceptible to recurrences.

Bacterial contamination of milk and water is a source of infection but one which is growing less in civilized communities. In this country the experience of New York City has been of great educational value. The total number of infant deaths in that city has been decreasing in spite of the growth in population. Life has been made more secure by the use of milk which has been kept clean and iced, being available in the home or dispensed from infant welfare stations to the poor of the city. The mother likewise receives instructions in the care of the food and of the infant. It is the experience of all who treat children that few cases of diarrhea now occur in the children of families that have been given instructions by the practicing physician or by the welfare stations. The production of certified milk has been of great benefit to childhood, not the least of which has been the educational influence in improving the quality of the general milk supply.

A considerable amount of dirty milk is still available, chiefly obtained from grocery stores of the poorer class. Epidemics of vomiting and diarrhea have occurred which affected all children using milk from one source, probably due to the presence of bacteria in dirty milk cans.

Milk with abnormally high bacterial count especially of pathogenic types has an influence in causing diarrhea in children under two years of age, if the milk be given unboiled. The New York Health Department and the Rockefeller Institute conducted a three-year experiment, one of the problems of which was to determine the effect of milk bacteria upon the child. When the number of microorganisms reached or exceeded one million per c.c., diarrhea was apt to be present. Bowel disturbances occurring in infants who were taking milk of less bacterial content seemed to be due to other causes as much as to the milk. The attitude of the physician should be that large numbers of bacteria in the milk are harmful, that dangerous organisms may be present from careless handling and from contamination, that milk should be produced and stored under the most cleanly methods and that all milk should be boiled and then iced while awaiting the infant's use. The feeding of raw milk, specially in the summer, may be dangerous; the best method is to boil the milk as soon as it is delivered to the home.

After the age of two years, diarrhea is not common from the usual bacterial contamination of milk, most cases being due to a specific type, the dysenteric bacillus, found in the late summer and early fall months. Many other sources of intestinal infection and injury are offered to the runabout child. Furthermore the intestinal wall gradually acquires resistance to the ordinary forms of bacteria introduced by milk.

The effect of external heat is of next importance. This may be due to the hot weather of summer, or to the overdressing of infants at any

time of the year. The incidence of diarrheal diseases is much greater in the summer months. Holt and Howland speak of the stagnant air in the homes as being a factor. Undoubtedly this plays a part in the tenements, and during seasons of the year when no ventilation is introduced into the room. Lack of fresh air influences first the nutrition and resistance of the child, secondarily favoring the development of diarrhea. In certain periods of the hot weather, it is impossible to find a cool spot either by day or night, and the only protection for the child is by frequent bathing, removal of all clothes and the use of the electric fan. The Chicago Infant Welfare Society through its physicians found that diarrheas in the summer months were more common in the children that wore too many clothes. DeBuys and others have studied the effect of humidity upon the causation of diarrhea in infancy. Its effect is not thus far known, but from the discomfort and oppression produced in high temperatures with marked humidity, the combination of the two undoubtedly affect infants as well as adults. Although diarrhea occurs in epidemic form at other seasons of the year, most cases are in summer. Warm weather must therefore be regarded as the most potent factor in the cause. This may act through causing lowered resistance, by decreasing the digestive juices and the urinary secretion, or by permitting bacterial changes in the food.

Chronic diarrhea may result from the injury produced by the acute or repeated attacks. Dysentery or inflammatory diarrhea is due to contamination of the food with specific organisms, and is a seasonal disease.

A classification which seems practical will incidentally be based upon the nature of the food, whether the child is breast or artificially fed, and upon the clinical symptoms.

1. Dyspepsia  $\left\{ \begin{array}{l} \text{in the premature infant} \\ \text{in the breast infant} \\ \text{in the artificially fed} \end{array} \right.$
2. Acute intestinal indigestion
3. Acute inflammatory diarrhea (ileocolitis, dysentery)

#### DYSPEPSIA IN PREMATURE INFANTS

The problem of greatest concern in the care of the premature infant is that of insufficient nursing and the intolerance for adequate amounts of food. The digestive tract must be regarded as the most vulnerable part of the premature organism. The tendency is towards an insufficiency of the processes concerned in the handling of food. The requirements for nourishment are greater because of the lessened production and greater dissipation of heat, the latter being due to the relatively larger body surface in proportion to the weight of the premature infant. Usually the child receives too little food and fails to gain, or if adequate amounts are attempted, intolerance and indigestion occur.

Premature infants may be perfectly healthy, especially as they approach full term, and if they have a plentiful supply of body fat. In such instances the chief evidence of prematurity is in the small size and body weight. The problems of successful feeding and survival of this type can be met by simple and intelligent care.

The poorest outlook for life is found in the debilitated infant who as a result of maternal or hereditary disease is born far ahead of the expected time and is markedly immature. However, disease and weakness may be present at birth in the child who more nearly approaches full-term gestation. In general one can regard the cases as less hopeful the shorter the period of intra-uterine life, but debility from incomplete development of the body tissues and structures and injury from unfavorable prenatal influences are of equal, if not greater, importance.

The functional difficulties offered by the immature digestive tract vary with the degree of frailty of the child. As regards the intake of food, there is usually no disturbance in the vigorous premature child. In the weakling there may be an absence of the suckling reflex, so that there is no attempt to nurse, or the effort is so feeble that the force is insufficient to extract milk from the breast. Frail premature infants do not swallow well, because of the weakness of the muscles of deglutition. The appetite is impaired or absent in certain periods. The vital processes are often too low for appetite to be present. Usually there is a tendency to sleep at the expense of eating, and it is with difficulty that the child can be aroused sufficiently to begin or continue suckling. The child tires easily while nursing and soon stops from exhaustion. As a rule, the greater the degree of prematurity, the less the desire for food. Those weighing as little as  $2\frac{1}{2}$  to 3 pounds are usually unable to nurse at all, or fail to do so because of constant sleeping.

The motor function of the stomach and intestine is likewise deficient, due to the weakness of the musculature. This has an inhibiting influence upon the secretion of the digestive juices, but there is also incomplete development of the secretory glands. The enzymes are present but are insufficient in quantity or concentration for the digestion of the relatively large amount of food needed, especially when the type of feeding is other than breast milk. The small capacity of the stomach is a factor which in a mechanical way interferes with nourishment, being insufficient for adequate intake, or showing intolerance by vomiting of excessive amounts.

There is a persistence of the fetal structures of the liver, and its function is therefore imperfect. Premature infants usually show marked jaundice. The small amount and late appearance of meconium are in part due to deficient secretion of the biliary and intestinal juices.

The subnormal temperature and the lowered metabolism play a rôle in the disturbed gastro-intestinal function.

Summarizing, one can say that the digestive ability will usually depend



upon the extent to which the premature approaches the normal full-term vigor and development. There are many conditions both within and outside the body influencing the digestive capacity, and these have a more disturbing effect on the debilitated child than upon the vigorous one.

In determining the capability of the individual premature infant for handling food, not only the period of gestation and the body weight must be taken into consideration, but some of the other features of incomplete development. It should be remembered that the commonest causes of spontaneous premature birth are maternal eclampsia and syphilis. Their effect upon the child is injurious and increases the hazards for living and the integrity of the body functions. Such infants have already suffered in their nutrition and have little subcutaneous fat and a much-lowered resistance. They may be toxic, or if not already infected are particularly subject to infection. Premature infants are especially liable to develop thrush and respiratory diseases. Physical deformities such as cleft-palate are greater handicaps than in the vigorous child.

How shall one conclude that an infant is premature? The reckoning of the mother as to the period of gestation does not always agree with the developmental appearance of the infant. More than once in prematures as low as 3 to 5 pounds, I have known the parents to maintain that the pregnancy had been full-term. It is more reliable from the clinical standpoint to judge from the signs of insufficient development. The estimated age at birth is of less importance than the health and vigor. There is no doubt about the prematurity when the weight is below 4 pounds, and it is usually correct to assume that a weight below  $5\frac{1}{2}$  pounds (2,500 grams) is an evidence of premature birth.

The pathological conditions arising in the digestive tract are with difficulty determined during life. The tendency to hemorrhage which is characteristic of prematurity, may affect the entire alimentary region. Bleeding may occur from the mucosa of the esophagus, stomach and duodenum, and appear in the stools. In the large intestines, hemorrhage is less common. Hemorrhage in the intestinal wall may result in lowered local resistance, infection, necrosis and peritonitis. Inflammatory changes in the mucosa are not infrequent and are productive of diarrhea. Starvation is imminent in weak infants who do not retain food. Inanition fever develops in some cases, though it is difficult to prove that an elevated temperature is due to such a cause. In prematurity the temperature rises easily from extremes of external heat in the incubator or warmed bed, or during extremely hot weather. The insufficient intake of water and milk so commonly occurring in these infants may be a factor in the deficient heat regulation.

The most common and at times the most severe digestive manifestation is vomiting. This may be due to feeding beyond the capacity of the stomach. Overfeeding is not probable from the small amount which the infant nurses, but may easily occur when food is administered by the tube. The



danger from vomiting is in the progressive loss of weight resultant from insufficient food retention. Regurgitation of food into the larynx will cause asphyxia.

The food requirements of these infants are so great as to cause almost complete absorption from the intestine and there is left little fecal residue. The stool is therefore small, and of the starvation type. It is usually brown and contains mucus. The infrequency is due to atony of the intestines and the lack of peristaltic stimulation.

Loose frequent stools are common. These may appear when the mother's milk "comes in," with its large amount of colostrum. Several yellow movements daily need not cause serious concern if they are digested and a steady gain in weight occurs. Looseness becomes pathological when there are also vomiting, infection and fever. Acute indigestion is of common occurrence, and the digestive disturbance is indicated by the curdled, bubbly, irritating character of the frequent movements. Blood is sometimes present.

The nutrition which in the frail premature infant is congenitally poor, is still further reduced by the underfeeding and digestive disturbances. The muscles are flabby, the fat scanty, the hemoglobin low because of the congenital deficiency. The skin is pale. The hemoglobin and red blood-cells increase as the weight improves, usually by the fourth or fifth month. Rickets develops early in the infants that survive. The tendency to convulsions which is common in the premature baby, is probably dependent upon the coexistence of rickets or tetany.

**Treatment.**—The prevention of gastro-intestinal disturbances lies in the use of all the careful methods necessary for the welfare of such a frail infant. The digestion in these infants will be aided if the body temperature can be maintained at the normal. In favorable cases this can be accomplished and requires only simple methods. The child should have a minimum of handling to prevent exposure, and should be kept wrapped in a cotton quilted jacket. An incubator is not necessary, and is only safe when constantly aired and guarded against overheating. A warmed open box or basket, which is available anywhere, is now generally used. The temperature of the environment need not be above 90° F.

The feeding is not difficult in the baby who is strong enough to nurse well. Breast milk is an essential for saving the frail infant. No effort should be made to use the bottle. If direct breast feeding is impossible, drawn breast milk should be given in small amounts every two hours after birth; this may be done without markedly disturbing the child. If swallowing is possible, small amounts of milk are introduced by a medicine dropper, not over a teaspoonful at a feeding until the tolerance is determined. It is not necessary for the infant to be awake. When the child does not swallow food, tube feeding is necessary, using a No. 12 French catheter and the barrel of a glass syringe. The amount which should be given is allowed to run into the stomach by gravity. It is not necessary or advisable to feed by

tube oftener than every four hours. As soon as the child is strong enough to nurse for a few minutes, this should be begun. When no milk is obtainable from the mother, breast milk should be secured from a donor. It is remarkable how readily milk will be furnished by some friend of the family or even from strangers. When the source of it is from a woman who has not had a Wassermann test, the milk should be brought to the boiling point. Tube feeding may be supplanted by the dropper, small spoon or a glass syringe with a rubber bulb on the upper end. Water should be fed in similar amounts by any possible method. Great care should be used in passing a tube, for the weak infant may have the mucous membrane injured, the stomach dilated or the respiration halted.

When no breast milk is to be had, and this occurrence is rare, small amounts of predigested milk, such as the chymogen milk recommended by



FIG. 33.—QUILTED COTTON JACKET FOR THE PREMATURE INFANT

Julius Hess, should be given. In recent years powdered protein milk has been used successfully, and in my experience has been found to be well handled. It should be fed in the same amounts as breast milk. The strength may be increased as the tolerance for it indicates. The following method of preparation is suggested:

Place 4 ounces of boiled water at 100° F. in a cup, sprinkle a level tablespoonful of the powdered protein milk upon the surface of the water, and stir with an egg beater. To this quantity add 2 teaspoonfuls of corn-syrup or dextrimaltose No. 1 or 2. This should be kept on ice and the quantity needed at feeding time be gently warmed.

The premature infant requires more food per pound of weight than does the full-term child. From 50 to 60 calories are needed for each pound. It will not be possible to feed this much at first, but it may be attained gradually.

Vomiting is difficult to control. One hesitates to use lavage in such a weakling. Water may be substituted for every other feeding. It may also be given under the skin. If minute quantities of breast milk cannot be

retained, it will be necessary to discontinue all feedings until the stomach becomes tolerant.

Hemorrhage from the bowel or elsewhere calls for the subcutaneous injection of at least 20 c.c. of whole blood, once daily. Diarrhea can be treated only by the temporary reduction in the amount of food, and the administration of water in any way possible.

#### DYSPEPSIA OF THE BREAST-FED INFANT

Dyspepsia, as a term used in infancy, refers to an acute or protracted disturbance in the digestion characterized by excessive fermentative changes in the food during its presence in the bowel. It has been called intestinal catarrh. The stomach is not particularly involved, except that food may be regurgitated from the accumulation of gas, and the gastric walls and circulation may be temporarily affected by the distention which adds to the paroxysmal pain.

**Etiology.**—Dyspepsia in the breast-fed child is commonly the result of continued overfeeding. The usual factor is the large amount received from plentiful breasts at too frequent or irregular intervals. In some cases the overfeeding may be due to an unusually high percentage of sugar in the mother's milk, an element which varies much in different women. As to the influence of the percentage of fat, it is difficult to say. Certainly in frequent and irregular nursings the percentage in some feedings will be higher than in others. The strippings from the breast were found in one case to contain 15 per cent of fat. The absolute quantity of the fat in the excessive amount of milk received may exceed the infant's tolerance, and may play as important a part as the sugar itself. In addition to the excess of these elements, it is possible that the high chlorid (salt) content of certain milk may cause frequent stools, as found in cases studied by Sisson and Dennis.

It is a common practice to nurse the baby for twenty or thirty minutes whether it desires it or not. In this connection, it is well to recall the observations of Charles Hendee Smith, who found that the breast infant gets practically all of its milk in the first five to seven minutes. It is difficult therefore to effectively reduce the amount taken at a feeding unless the time be limited to three or five minutes. Longer intervals offer the best method.

Dyspepsia in the breast-fed infant can sometimes develop as the result of insufficient food. Marked hunger causes excessive contractions of the stomach and intestinal walls. There has been found to be a decreased amount of gastric acid and intestinal juices during starvation, allowing increased activity in bacterial growth and the production of diarrhea. Starvation, if prolonged, aids in the development of marasmus. Much the same symptoms are present in underfeeding as are found when too much breast milk is given. The crying, nausea, vomiting and colic are evidences



of the discomfort which the infant undergoes. Incomplete nourishment soon causes weight loss, weakness and either a lack of bowel movement or diarrhea. This insufficiency of food is not at all rare and is easily determined by weighing.

**Symptoms.**—The characteristics of dyspepsia are found in the two illustrative cases which follow :

Case 1.—A newly born infant at ten days of age was having eight or ten loose stools daily. The child was somewhat uncomfortable, and the weight had shown marked increase over that at birth. This with the nursing at three-hour intervals day and night suggested the plentiful supply of milk obtained. When the infant was weighed before and after each nursing for twenty-four hours, it was found to be getting from 4 to 5 ounces each time, making a total in excess of 32 ounces. As rarely happens the milk was even more abundant after 6 P.M. Improvement followed the lengthening of the intervals to four hours, the limitation of the time at the breast to ten minutes, and the withdrawal of one night feeding. The stools did not become entirely normal until the nursing time was reduced to five minutes.

Case 2.—A breast baby, aged two months, who had become overweight had always had too many stools, as high as twenty daily. The movements were thin, highly acid, mucous, with numerous small white curds. At times the feces spouted from the anus, at other times the character was more nearly normal. The infant cried night and day, and seemed to have much pain from tenesmus and colic. The skin of the napkin region was raw. The urine was not increased in amount, but contained sugar, evidently of alimentary origin. The cause of it all was found when the nursing history became known. The mother had always nursed the child every time it cried. This resulted in almost continuous feeding, and much milk was obtained. This overfeeding had gone on since birth, and the child's discomfort was becoming worse.

Reduction of the nursing period to ten minutes and an increased regular interval of four hours did not materially diminish the number of stools. When the breast feeding was held to five minutes and in addition a few ounces of undiluted skimmed boiled milk were fed, improvement began. As long as the mother continued to nurse, the additional food was necessary to control the excessive fermentation and number of stools.

In long-continued overfeeding and diarrhea the mucous membrane undergoes superficial inflammatory injury from which it requires much time to recover. It is probable that as a result of vomiting and of wastage from too numerous stools, the child is actually hungry. This will be the case in the child with stationary or losing weight. The discomfort, colic and screaming are apt to lead to still further overfeeding, by disregarding all attempts at regularity.

The nutrition may not be affected in the cases of short duration. Variability in the weight is the rule. It is remarkable that early in the disturbance the infant may become overfat in the presence of excessive stools. When the condition of dyspepsia is long continued the child remains stationary or shows a net loss of weight. The muscle tone is below normal. The skin becomes pale, and perspiration is active during periods of colic.



Many such infants have an exudative tendency shown in the appearance of facial eczema and a seborrheic crusting of the scalp.

**Diagnosis.**—This should be easily made in the breast-fed infant having persistent diarrheal stools. Overfeeding should be at once suspected, and determined by careful weighing before and after feeding for one or two days. An accurate pair of beam scales is necessary for this. Such a procedure is as valuable in determining overfeeding as it is in underfeeding. The character of the crying is somewhat different in underfeeding, and colic is not the cause. Hunger stools are small, mostly mucous, having little food material and no fermentation.

The hypertonic or nervous infant comes first into question when colic is present. Such an infant has constipated as well as diarrheal intervals. The stools may be dyspeptic at times, and the child may be overfed in the effort to stop the crying. Atropin in sufficient dosage is a diagnostic as well as a therapeutic agent. Spasm and hyperperistalsis of the stomach and intestine are visible. The distinction between the two is best made by the character of the stools which are more numerous, larger and more acid in dyspepsia. In some instances the separation of these diseases may be impossible.

Infections of the upper respiratory or of the urinary tract will cause indigestion and paroxysmal pain in certain infants. These attacks are of short duration, are febrile, and are in contrast with the ordinary state of the child's health. There is no fever in the uncomplicated case of breast-milk dyspepsia.

Intussusception, because of its occurrence in infancy, and the urgency in its diagnosis, should be thought of in acute pain. The acuteness of its onset, the presence of blood in the stool, the freedom from previous vomiting and colicky attacks, the development of obstruction, tumor, peritonitis and shock are characteristic of intussusception. The same causes which produce invagination in other infants may cause it in dyspepsia, so that it should always be borne in mind.

**Treatment.**—It would seem that the handling of such cases is obvious and easy. But this is far from the fact. In many cases seen early and with the coöperation of the family, the results are strikingly prompt and permanent. In others they are tedious and may wear out the attendants and especially the physician.

The dyspeptic and colicky infant is apt to be weaned instead of regulated. This misfortune should be prevented if possible. Only as a last resort should the breast be entirely discontinued.

The information that the child must be fed less milk meets some opposition from the attendants. It is well for the physician to emphasize that the infant gets all it needs in from five to ten minutes at the breast, that four-hour intervals will decrease the number of stools, that as soon as diarrhea ceases the child will be better satisfied, and more comfortable.

If diarrhea and colic continue other dietary measures must be added. The five-minute nursings must be complemented by the feeding of a high protein, low fat and sugar addition. One of the simplest is the solution of a casein powder in water, 1 level teaspoonful to 2 ounces of water, sweetened with saccharin  $\frac{1}{10}$  grain. Calcium caseinate is obtained as protolac, casec, larosan, etc. This should be given at the end at each nursing. The commercial protein milk powder can soon be substituted for the foregoing. This can be given in the proportion of 1 level teaspoonful to each ounce of water, and sweetened with saccharin only. The infant may take 2 or 3 ounces with each nursing to good advantage. Fat-free buttermilk or boiled skim milk is usually well tolerated. These foods not only check the fermentation, but supply the water which the child needs.

Gastric lavage and intestinal irrigation may relieve the vomiting and the distention, and are indicated in the beginning of the treatment.

Anodynes are seldom needed. A teaspoonful of castor oil on the first day may give more comfort and rest than the infant has had for some time, especially if the feeding is thereafter reduced as above mentioned. There is no objection to the administration of ten drops of paregoric once or twice daily until the conditions are made easier. I have never resorted to the usual household remedies for colic. Adaptation of the diet to the infant is the first and chief concern, during which the simple methods above described are employed.

**Prognosis.**—The prognosis is favorable. The condition is never fatal. Some infants may never get formed stools so long as breast milk is continued. They may likewise be susceptible to diarrhea throughout early childhood, as occurred in one of my cases.

#### DYSPEPSIA OF THE ARTIFICIALLY FED INFANT

**Etiology.**—This type of indigestion is dependent upon numerous factors. The underlying cause is the frequent inadaptability of many artificial foods for the digestive tract of the young human species. Although fortunately it has always been possible to successfully rear many infants on food other than the breast, and the percentage of these is increasing, it is much easier to exceed the tolerance in artificially fed infants. Such a digestive disturbance is more serious and is harder to control.

While breast milk varies somewhat, the percentage of its elements and the total amount secreted by one mother are within limits which are reasonably safe. With artificial feeding we are dealing with food which is foreign to the young infant, and in which the proportions or the quantity fed may be unsuited to the digestion of the individual. It is now generally recognized that the overfeeding with sugar and fat, either one or both, or the excessive amount of milk as a whole is responsible for the production of digestive intolerance. In this connection should be mentioned the valuable

work of Marriott who has shown that the indigestibility of cow's milk may be due to the high buffer value of the salts which exceed the capacity of the gastric acidity to neutralize. For this reason the feeding of soured milk mixtures is much better tolerated.

Overfeeding is a relative matter. The infant with congenital weakness or inferiority does not handle food in the quantities taken by the robust child. Any infant will show less desire and less tolerance during hot weather. Acute or chronic infections, whether within or without the digestive tract, and poor nutritional states reduce the digestive ability.

Previous diarrheal attacks and the resulting nutritional failure make the infant more vulnerable.

Infants with beginning nutritional disturbance characterized by stationary weight and known as dystrophy or hypothyrepsia are especially susceptible to attacks of diarrhea. The fat constipation which characterizes this nutritional state is succeeded by a fatty diarrhea when excessive sugar is added to the diet.

**Physiological Chemistry.**—Little is known about the chemistry of this type of indigestion. The changes occur in the duodenum. The excessive fermentation is produced by acid-forming bacteria in splitting the large amount of lactose or other sugar which has been ingested. The fat is likewise incompletely split, and there is much free fatty acid, which proves irritating to the intestinal lining. The alkaline (duodenal) secretions are poured out in the attempt to neutralize these acids, peristalsis is stimulated and stools of a watery consistency, high acid content and greater frequency result. Excess of fatty acids remains unneutralized, and the fatty soaps which are characteristic of formed stools are much diminished in this disturbance.

**Pathological Changes.**—There are no important intestinal lesions in simple dyspepsia, so far as is known.

**Symptoms.**—There are no severe general symptoms in the usual case. In the infant who has been uncomfortable and not gaining, there may be preceding symptoms of poor appetite, regurgitation of food, and flatulence. Vomiting usually initiates the attack of indigestion. In the previously well infant who has been having a smooth, soft or formed stool, the movements become changed to markedly loose with much water loss. The odor is sour and at times offensive, the color green, and the stool contains mucus and undigested particles, probably due to rapid passage through the bowel. The reaction is always acid, irritating to the buttocks, and within a few hours may cause excoriation. Erythema and usually inflammation of the skin occur under the diaper due to irritation from the highly acid stools. There are frequently patches of excoriation and superficial denudation. There is straining from the increased peristalsis and much discomfort and colic from the intestinal gas. The sleep and rest are disturbed. The infant cries much

and is irritable. There is little if any fever. Stomatitis is not infrequent during diarrheal attacks.

Relief from diarrhea is followed within a few days by general improvement in the lighter cases, properly treated. The stools gradually become formed, and after the appetite returns a gain in weight slowly occurs. This is the common outcome of the private case in the home.

It is common, however, in the institutional child to see long periods when the stools are not normal, hardly loose, but full of mucus and much undigested material. If the child be left in the hospital or exposed to respiratory infections there will soon be a repetition of the diarrhea, and with each attack the severity increases and the convalescence is more tedious. The nutrition becomes definitely impaired. Such cases may pass into the more severe type of indigestion, and develop toxicosis, or enter a state of athrepsia.

**Treatment.**—Dietetic treatment, it should be emphasized, is of the most importance. No relief will be obtained until the food is changed. An initial teaspoonful or two of castor oil will aid in the elimination of the irritants, especially if the stool be foul. In acute primary indigestion, the food should be withheld for twenty-four hours, giving instead weak tea or water sweetened with saccharin. Because of the small amount needed to sweeten tea or water and because it has a somewhat sickening taste, less saccharin should be added than is used in the sour-milk feeding which is next begun. Sour milk without fat or added sugar is digested with little formation of fatty acids in the intestine. Churned buttermilk is available in the country and small towns and should be diluted with gelatin or barley water. If skim milk be used it should be boiled, cooled and then soured with lactic acid organisms, such as the Bulgarian bacilli. The constipating and nutritional value of the food is still further increased by the addition of dried casein or the curd from boiled skim milk. Within two days of beginning these foods, it is well to add corn syrup or dextrimaltose (No. 2)  $\frac{1}{2}$  ounce for the day's feeding, and to increase this cautiously during the next few days. The use of the dried protein milk now obtainable from any druggist is to be recommended, beginning with half the usual strength, and then increasing to the standard concentration (10½ level tablespoonfuls to a quart of water). The above-mentioned sugars should be added in the same careful way.

In the poorly nourished infant, such as infantile atrophy (marasmus), no starvation periods should be considered. It is, however, just as important that overfeeding should be avoided, because of the tendency to exaggerate the dyspeptic symptoms. The feeding should be protein milk, without added sugar, 1 ounce of the regular mixture for each pound of body weight as the twenty-four-hour requirement, and enough added water to supply the infant's needs. Each day as improvement takes place, this may be increased until 32 ounces are given, and malt sugar gradually added



up to 1 ounce daily. In these cases breast milk given in small amounts in combination with protein milk is of great benefit, as both of these are detoxicating foods. The infant should be kept from exposure to infections, and if in the hospital should be taken home where he may have the advantage of fresh air and sunshine.

The burns from acid stools may be prevented, in part at least, by prompt cleanliness in removing soiled diapers and washing with warm water without soap. The treatment is first the reduction in the number of stools to the normal, and the relief of the diarrheal and highly acid nature of the stool which occur in the general treatment of the disease. The diaper should be left off entirely when "scalding" occurs, and a fresh pad frequently put under the buttocks. Keeping the skin dry from further contamination and by air is the best of all treatment. In severe cases the protection by melted tallow or paraffin is the best application.

#### DIGESTIVE INJURY FROM STARCH

A dyspeptic and nutritional disturbance to which the attention of the practicing physician needs especially to be called is that designated by Czerny as a starch injury. It has happened in my practice and to my knowledge in the experience of others. The occurrence is brought about usually in this way: during a vomiting or diarrheal upset the infant is taken off milk and placed upon a gruel, such as barley, rice or wheat. Instead of its temporary use for one or two days, the cereal alone is continued indefinitely, often for weeks. Excessive fermentation results from starch indigestion, the functions of the intestinal tract are impaired and a relative starvation results. Attempts to increase the dietary show that not only is there an intolerance for starch but that cow's milk digestion is disturbed. In other cases milk and salt are added in too small amounts and the starch in excessive quantity.

Starch, well-cooked, is usually well digested by infants when given in suitable amounts, if combined with milk. When cereal is excessive and forms a one-sided diet, there results a food deprivation, because of the deficiency in fat, protein, salts and vitamins. It is a common experience to produce rickets in experimental animals by an exclusive cereal diet. The injurious action of the starch is probably through its affinity for the water of the body tissues, resulting in desiccation, diarrhea, and abdominal distention. When salt is added, edema develops and the retention of water causes sudden increase in body weight.

**Symptoms.**—The injury may begin at any age in infancy if cereal alone is fed for too long a time, or if in too large a quantity relative to the amount of milk. This is most apt to occur during or after an acute gastrointestinal disturbance.

The stools are sour, thin and watery, slimy, yellowish-brown and too frequent. Starch may be found by the iodine test. The abdomen becomes

sunken, the subcutaneous fat disappears, the weight decreases and the muscles develop a doughy consistency. The skin is dry, pale, pasty, and the child becomes dried out. The eyes show an abnormal dryness (xerophthalmia), cloudiness; vesicles and inflammation are apt to develop. Rickets is not an infrequent accompaniment, with the well-marked beading of the ribs, thickening of the wrists, and sweating of the head.

As a result of the lowered resistance to infection, furunculosis, otitis media, lesions of the mucous membrane of the mouth, and respiratory diseases usually complicate the digestive and nutritional disturbance.

In one type, known as the hypertonic, the muscles are rigid, the spinal column stiff and the infant lies with the head retracted.

The prognosis is bad in infants under six months of age, who may succumb. The difficulty in feeding is not only the intolerance for starch, but for other carbohydrates, and also for milk, so that the digestion regains its function slowly, if at all. The infant is fed ordinary formulæ without success.

**Treatment.**—These infants need to be put upon breast milk for several weeks or months. Gradually small amounts of undiluted acid milk are substituted for the human milk. Buttermilk or concentrated protein milk are the best preparations. Later sweet skim milk, vegetable and meat broths and orange juice should be added to the diet. No starchy food should be tried.

Furunculosis should be treated by drainage of the pustules, antiseptic applications, and, if persistent, by vaccine therapy.

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## CHAPTER XIV

### THE INTESTINES (*Continued*)

#### DIARRHEA (*Continued*)

##### ACUTE INTESTINAL INDIGESTION

This type of intestinal disturbance, occurring chiefly during infancy and the early years of childhood, is a common clinical manifestation. Symptoms of acute indigestion and more or less evidence of constitutional disturbances characterize this diarrheal disease. Severe cases cannot always be clinically differentiated from ileocolitis, but the intestinal lesions are mild and superficial as compared with the severe inflammatory and ulcerative changes in dysentery.

The disease is more common in the summer months due to various factors concerned with hot weather. Gross overfeeding at any age is more dangerous during the summer. Debilitation of the child on hot days or during long periods of heat undoubtedly plays a part in the causation. This acts by reducing the amount or the activity of digestive secretions and possibly the gastric motility. As a result the food is undigested and excessively fermented in the intestine, is incompletely absorbed, and, through irritation of the intestine, increases the peristalsis. Spoiling of the milk or other foods is also more common in warm weather and must also be considered as one of the factors. Decomposed and contaminated foods introduce not only microorganisms but their toxins as well. Cases of paratyphoid infection should be separated from the class under discussion, as these are epidemic and are due to a positive bacterial or toxic cause.

Many foods known to be difficult of digestion are taken by infants and young children, resulting in acute intestinal upsets. Unripe fruits or incompletely cooked vegetables are common examples. While these act mechanically they may be followed by severe and toxic disturbances. Increased susceptibility to intestinal indigestion is present in the frail child. Improper artificial feeding and resulting malnutrition with repeated attacks of indigestion predispose to further digestive disturbance. Infants fed solely on the breast are not subject to severe diarrhea.

Acute intestinal indigestion may occur as a result or accompaniment of infections elsewhere in the body. Such parenteral diseases during the presence of fever may act similarly to heat in reducing the digestive secretions or they may produce damage by their toxins. Infections are especially

frequent in bottle-fed infants, particularly in institutional children, during the seasons when respiratory diseases are common. I have repeatedly seen infants who had been digesting food well, develop vomiting and diarrhea when epidemic rhinitis, otitis media and other upper respiratory infection occurred. Recently Marriott and Jeans have called attention to the importance of latent or hidden mastoiditis and paranasal sinusitis as causes of cholera infantum. Undoubtedly such infection may cause, accompany or follow intestinal indigestion and intoxication.

It is generally believed that the origin of diarrhea is in the duodenum. One may ascribe the inception of the disease to stasis whereby more favorable conditions are present for the excessive growth of the normally present fermentative organisms. Pathogenic organisms may be introduced by way of the mouth. While no organism has been proven to be responsible for the disease, and although the types and numbers found in diarrhea are numerous, little is as yet known about the part played by bacteria. The colon bacilli, streptococci, dysentery bacilli and numerous other microorganisms which have been found in the stools by some observers may be only secondary invaders and may owe their development to the favorable conditions present in the intestine.

Irritation of the intestinal mucosa, chiefly by the fatty acids, increases water extraction from the gut and stimulates peristalsis, thereby resulting in frequent watery stools.

The blood becomes concentrated due to the body depletion from fever, starvation, loss of much water and digestive juices. Much mineral salt is excreted in the loose stools, so that little sodium chlorid and calcium are retained. The blood shows no chemical disturbances in the mild cases, but when intoxication develops it is found that the waste products of metabolism are greatly increased. Schloss reports the total nitrogen retention in the blood to be increased as high as 233 milligrams (the normal 30 to 40 Mg.), and the urea nitrogen as high as 108 milligrams (normal 14 to 20 per 100 c.c. of blood). He ascribed this increase to the impaired function of the kidney and its faulty elimination of waste products. The urine is greatly reduced in diarrheal and intoxicated children.

The intestinal lesion is regarded as a catarrhal enteritis, an inflammation limited to the mucosa and its glands. At autopsy such evidences may be found in the ileum, cecum and colon, but frequently no anatomical lesion, with the exception of congestion, is found.

The infant that dies with intoxication shows constant fatty changes in the liver, degeneration (cloudy swelling) of the kidneys, and a distention of the intestines, with areas of inflammation of the mucous membrane or a denudation of the epithelium. Usually by microscopic examination the lymph follicles show cellular proliferation, and surrounding the blood-vessels are areas of leukocytic infiltration and petechial hemorrhages.



**Symptoms and Course.**—Three principal types of the disease are seen. These are the mild cases, the fulminating cases known as cholera infantum, and more prolonged severe cases with or without acid intoxication.

In the *mild type*, seen usually in older children, no gastric symptoms are present with the exception of temporary vomiting in the case of sudden onset. Diarrhea soon appears, the movements becoming four to five times the normal number, the color green, the character watery and often showing fermentation. At first there are undigested remnants of food, and later great increase in the water and mucus. The odor depends upon the predominating character of the recent diet. If sugar and fat are still being given the odor is sour; if the diet is chiefly protein there is a putrid odor. The reaction to litmus paper is usually acid. There is no blood present in the stools. The urine is decreased. The child is restless, may cry from pain, and the abdomen may be distended.

The temperature is high at first but in two or three days settles down to the normal if the child's feeding is reduced. Convalescence is rapid in the properly treated vigorous child past the age of infancy. In the weak infant the attack may be prolonged, and following periods of improvement there may be recurrences from which the nutrition further suffers. It is in such cases that the next type of diarrhea is occasionally seen.

*Cholera infantum* is an unusually rapid and severe type of diarrhea, dehydration and intoxication occurring in early childhood, and it is so named because of the similarity to cholera. The onset is abrupt with retching and obstinate vomiting. The vomitus soon consists of serum, bile or in some instances fecal material. The thirst is naturally great. The stools are little different from other severe types of acute diarrhea, but peristalsis is more violent and the movements are composed mostly of much serum, with a greenish stain and with membranous flakes or mucous shreds. The bowels move more frequently than in any other type, being almost continuous. The body weight decreases rapidly, due to dehydration. Prostration is a characteristic symptom beginning early. The temperature is high from the first, 104° to 105° F., and steadily rises to 106° or 107° F. Restlessness in the first hours of the disease, convulsions, and finally stupor or coma are the nervous manifestations seen commonly during the short course of this profound disturbance.

In most of the *severe cases* the course is not so rapid nor so violent as the preceding. The temperature however early becomes high, 104° to 106° F. Vomiting may be persistent. The watery stools may number ten to twenty in twenty-four hours. These become green or change to brown. The odor is at first offensive, but as soon as the bowel is empty of undigested food there is no odor or only a faint one. Large amounts of mucus may be present.

Diarrhea lasts five or six days, following which the case recovers if favorable conditions are present. The early treatment, the care with which

the feeding is managed, and the inherent vigor of the child are factors in the recovery.

When the disease persists, the child shows much restlessness and convulsions may occur. Stupor or collapse may intervene early or not until later in the course. The fever may remain high, seldom coming below  $103^{\circ}$  F. The diarrhea may show no tendency to lessen. The general appearance of the child is one of great prostration. The eyes remain partly closed, respond feebly to stimulation, the eyeballs are dry and covered with a film. The fontanel is sunken. The lips are red, the tongue is coated and at the tip and edges the papillæ are prominent and reddened, and the entire mouth dry. The skin is dry and ashy, the abdomen is flabby and sunken. The liver may be felt below the costal margin. The earlier restlessness becomes replaced by abnormal quiet, complete relaxation and a failure to notice the surroundings.

There is little excretion from the kidneys, and in many cases the urine contains sugar, probably of alimentary origin, due to increased permeability of the intestinal wall. Albumin and casts are commonly present, and chemical examination may reveal acetone and diacetic acid.

The fatal cases end in from three to seven days, usually in a convulsive or comatose state.

The picture just described is that of intoxication. Not all of the cases develop the severe manifestation which is recognizable as *acidosis*. The presence of acetone bodies in the urine is not sufficient for the conclusion that acidosis exists, for these may be found in cases of persistent vomiting and even in alkalosis. Acidosis is easily recognized by the air-hunger (hyperpnea) which the child shows. This exaggerated breathing is one of increased depth but is not increased in rate beyond that which accompanies a high temperature. Other characteristics of acid intoxication are the lowered alkaline reserve in the blood, and a decrease in the tension of the carbon dioxid expired from the lungs. The normal range for the carbon dioxid tension of the exhaled air is from 35 to 45 milligrams, but in acidosis it is often as low as 15. This lowered tension may be demonstrated by the colorimetric method of Howland and Marriott. The lowered alkaline reserve of the blood is shown by the increased acid concentration, determined by the method of Van Slyke. Acidosis is a symptom only, and is a serious one in its prognostic import in this disease, for although it may be caused to disappear by plentiful water administration and by alkalies, no relief from the underlying disease is obtained.

**Diagnosis.**—Attention has been called by Schloss to the resemblance in the symptoms of intoxication and uremia, the former being a purely functional disturbance due to water loss from the intestinal tract and to the diminished urine, the latter caused by organic disease of the kidney.

The existence of sugar, acetone and diacetic acid in the urine is suggestive of diabetes, especially if intoxication and diarrhea are also present.

The previous history and the determination of the amount of blood sugar will help in the differentiation at this stage of both diseases.

As has been mentioned an alimentary disturbance and intoxication may sometimes occur as a result or accompaniment of infection outside of the intestinal tract in infancy and early childhood and there may be many of the same symptoms with diarrhea, dehydration, deep breathing and fever.

The nervous symptoms are suggestive of cerebral disease such as meningitis and epidemic encephalitis, in which however diarrhea is usually absent.

I would call especial attention to the danger of overlooking intussusception. Just why the initial hemorrhage and the obstruction, cardinal symptoms of invagination, are disregarded, I can only explain by the failure to get a history of the early course of the illness and because the physician does not personally inspect the evacuation from the bowels.

**Complications.**—Secondary pyelitis is frequently found and should be looked for when there is a return of fever. The urine should be routinely examined during the course of any diarrhea. I have seen the appearance of non-specific vulvovaginitis with high evening temperature after the stools had become normal.

Stomatitis, nasal and ear infections, pneumonia, peritonitis and furunculosis may develop in the severely affected child.

A long period of invalidism or nutritional disturbance may follow the disease, and relapses are not unusual.

**Prevention.**—One sees few cases of this disease among children whose feeding and hygiene are carefully supervised, and this fact demonstrates the value of pediatric advice both from the private physician and the infant welfare station.

Children should be kept comfortable at all seasons of the year, but especially during hot weather by frequent baths, cool outdoor air, and suitable clothing. The food should be reduced and the water increased on hot days. Young children, especially frail ones, may be spared intestinal upsets if sent away where it is cool.

The physician should recommend the use of certified or other safeguarded milk. It is advisable to boil all milk and water given the infant or frail child. During the summer months disturbances may be prevented if the child be fed simply on wholesome table food, and not allowed to have those articles which any physician knows may be a source of gastrointestinal irritation.

Children may often be protected from infections by being kept away from crowds, and from exposure to children already ill. The hospital child should be sent out to an environment where the least danger of infection exists. Hygiene and fresh air are especially necessary in the care of institutional children.

**Treatment.**—The uncertainty at the outset as to whether cases will be mild or severe makes it necessary to begin proper treatment at once. When

vomiting and diarrhea appear all food should be temporarily withheld, and throughout the course reliance should be placed chiefly upon the importance of a properly restricted diet. If this includes the administration of fluids so as to replace as rapidly as possible the water balance of the body, few other measures are needed, especially in the mild cases.

It is now recognized by pediatricians that few drugs are indicated, and that most of those which have attained much vogue are probably useless. In this disease the vomiting and diarrhea rapidly empty the gastro-intestinal tract of undigested and toxic contents, so that purgatives are not needed. Their use may add to the discomfort, water loss, urine reduction and exhaustion. Drugs which are supposed to have an antiseptic or astringent action have not been found of value. Astringents should not be given early in the disturbance and they are ineffective later. Sedatives should be given for restlessness or convulsions.

While there is no specific for the cure of acute intestinal indigestion, a certain routine treatment is of assistance to the natural recuperative efforts. This is comprehended in the following measures:

For the first twenty-four hours of the disturbance and until vomiting has stopped no food is to be given. Sweet milk is to be indefinitely discontinued.

For the vomiting daily *lavage* with boiled water, Ringer's or normal saline solution, 500 to 1000 c.c. is of great benefit. A portion of this solution should be left in the stomach.

*Water* is to be given *by the mouth* frequently, but in quantities that will not be vomited. Cool water is better retained if drunk in small quantities, and is advantageously given with a teaspoon. In some cases shaved ice is relished.

In the children who continue to vomit and in whom the diarrhea is causing much loss of fluids, water must be given by other routes. There is no need to wait until the child is severely dehydrated before beginning such methods. In cholera infantum or other severe cases the child needs fluids at once, and this is most practically given by hypodermoclysis. A pint of saline solution will be taken up, injected subcutaneously every twelve hours.

If improvement is not seen by an increase in the urine and by the reduction of the diarrhea it is advisable to use intravenous injections of normal salt solution from 300 to 500 c.c., glucose solution 10 to 15 per cent, 60 to 120 c.c., or in case of air-hunger, fresh bicarbonate of soda 24 grains in warm sterile water, 60 c.c. These solutions should be injected slowly and repeated daily if necessary.

Sterile isotonic salt solution may be given once a day *intra-peritoneally* in amounts of 100 to 300 c.c. From 3 to 5 per cent glucose solution 100 to 200 c.c. may be employed, alternating with the saline solution. This



method is useful in infants, but if abdominal distention be present it is not a safe procedure.

Any of these procedures may be safely carried out by the physician in the home if a hospital is not available. A trained nurse will be of service in the preparation of the solutions, and in the care of the child.

The dietary will depend upon the age of the child and the seriousness of the disease. Infants may be given breast milk on the second day and later, alternating with small amounts of buttermilk or protein milk. Sugar need not be added to the sour milk while some breast milk is being fed, but it may be taken better if sweetened with saccharin.

In older children buttermilk or protein milk is the food of choice for the first few days, after which gelatin, rice gruel, beef broth and unsweetened orange juice are indicated. A more general diet will be employed as soon as the child is convalescent.

Prompt treatment of parenteral infections should be begun. If mastoiditis or sinusitis be present drainage is necessary.

For the nervous symptoms sodium bromid or chloral hydrate may be found of benefit.

Opium is not indicated in the first two days of the illness. It may become necessary to give it for relief of pain and continued hyperperistalsis. Paregoric and Dover's powders are the preparations usually employed. It is better to give an occasional dose than to make it an ingredient of a mixture given at frequent intervals. Opium should not be used when the child is comatose or if after administration for a few days the symptoms are not improved thereby.

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## CHAPTER XV

### THE INTESTINES (*Continued*)

#### DIARRHEA (*Continued*)

##### ACUTE INFLAMMATORY DIARRHEA

##### (*Acute Ileocolitis, Acute Dysentery*)

**Descriptive Summary.**—Acute diarrhea which is associated with pronounced inflammatory lesions of part or all of the thickness of the intestinal wall is a disease belonging especially to infancy and early childhood. It is due to bacterial infection chiefly with one of the dysentery group of bacilli. In contrast to the other types of diarrhea previously discussed, this disease is usually accompanied by blood-stained or purulent stools.

The course shows a tendency to be protracted, in which regard the appearance is not unlike typhoid and paratyphoid fever. In cases that live, the outcome is often attended by a long and incomplete convalescence. The mortality is high in children, and death is due to severe and progressive intestinal lesions, to toxemia, wasting and to terminal respiratory infections.

**Etiology.**—The disease is rare under four months of age, and it has been estimated that about one-third of the cases occur in the first year. Throughout the second year there is found a large number of cases, and although no age is exempt, the percentage steadily decreases after three years. Infants while being fed at the breast alone do not contract the disease. Young children are more susceptible because their intestinal tract is more vulnerable and the character and preparation of their food are often at fault. The opinion that the second summer is a dangerous period for this disease is true because there is often less care in the dietary and the child beginning to run about has more opportunity for contamination and infection.

Dysentery is more common in dirty, careless homes and in unscreened houses. Although sporadic cases do occur in private practice, there are often other members of the family who contract the disease by direct contact. Smillie reported twenty-one such instances. Epidemics in communities and in institutions are not infrequent. Carelessness in the disinfection of diapers and stools is the source of spread. Flies may harbor the infecting organisms and carry them to exposed food. The hands of the nurses or other attendants may remain contaminated. Since the dysentery bacillus is not found in the stools of individuals who have never had the disease

and no habitat outside of the body or its excreta is known, it is most probable that transmission comes from a common direct source. This may be from an active case, a convalescent or a persistent carrier. Through these, the water, milk, and other exposed food are thus contaminated.

There is a definite seasonal characteristic in the incidence of most cases, and this is seen in the late summer and in the months of September, October and November. The peak of the frequency is usually not in the hottest weather and so far as is known not dependent upon the atmospheric humidity. Epidemics may, however, occur at any time from the beginning of summer till late in the fall. The first case in the community probably originates from a carrier, and the development of other cases therefrom depends upon the failure to prevent the spread by contact and contamination. Kendall concluded that the type of dysentery microorganism prevalent in the fall months is carried over into the following summer, though the types may vary from year to year.

So many varieties of bacteria are present in the stools that it is difficult to determine the etiological relationship. Some may be of only minor importance as secondary invaders. The *B. dysenteriae* exists in numerous types, and some one of these is usually regarded as the direct cause of the disease. Kendall found the Welch bacillus, the Flexner and the Shiga bacillus as well as the *B. mucosum capsulatum*. Wollstein recovered the Shiga, Flexner or Mt. Desert types in twenty of eighty-six cases of diarrhea which she studied. Even in cases of dysentery with much blood and mucus other varieties of organisms may be more abundant than the dysentery bacillus. The streptococcus is frequently found and probably develops secondarily in the intestinal lesions.

**Lesions.**—The inflammatory diarrheas show definite changes in the intestinal wall, which vary usually with the severity and duration of the disease.

In general the pathological lesions are those of catarrhal inflammation of the intestinal mucosa, hyperplasia of the lymph-nodes, denudation of the superficial epithelium, and ulcer formation of various size and extent. It is generally considered that the pathologic course follows somewhat in the sequence just mentioned.

The term ileocolitis designates the usual area involved in the intestines. The disease may be confined to the colon, but this is rare. The changes in the ileum are usually confined to the terminal one-third. Infections causing enteritis may in infants affect all portions of the intestinal tract. The lesions are diffuse, especially in the epithelial cells lining the intestine. Okubo found that by injection of the toxins from dysentery into experimental animals there resulted a diffuse catarrhal enteritis involving the large and the terminal one-half of the small intestine.

Catarrhal inflammation of the intestine involves not only the solitary lymph-nodules and Peyer's patches which are grossly enlarged and hyper-

plastic, but microscopically there is cell proliferation in the mucosa. A superficial denudation is evident in certain areas. When ulceration of the lymph follicles occurs, a portion or all of the nodule may be involved in the necrosis. It is common to find over the mucosa an exudate of tenacious sero-bloody mucus.

Hemorrhage occurs chiefly in the catarrhal form of ileocolitis, and is not common from the localized ulcers. Ulcers seldom perforate the entire thickness of the intestinal wall.

The lesions of acute pseudomembranous dysentery are characterized by an accumulation of fibrin upon the mucosa, and an infiltration of the mucous and submucous coats of the ileum and colon. The membrane consists of fibrin, cells and bacteria. In some cases the colon undergoes severe suppurative inflammation, with superficial and sometimes deep ulcers.

**Course of Lesions.**—At the onset there is hyperemia of the mucous lining of the intestine, which secretes much mucus and may become tinged with blood. The swollen mucous membrane has numerous punctate and linear hemorrhages.

Small flecks of mucus adhere to the intestinal folds under which there is necrosis of the superficial layer. The child may then recover, but the lesions usually go on to definite ulceration. A thick layer of exudate covers the folds of the intestine and is connected with the underlying necrotic mucosa. When this superficial layer comes off, various-shaped ulcers are found, occasionally deep, and extending into the submucosa, muscular or subserous coats. Ulcers may be localized, or may spread and coalesce, from which the child may pass large shreds of the coagulated mucous layer. Portions of the intestines show edema, inflammation and hemorrhage.

As the ulcers heal granulation tissue is produced, and the new layer of

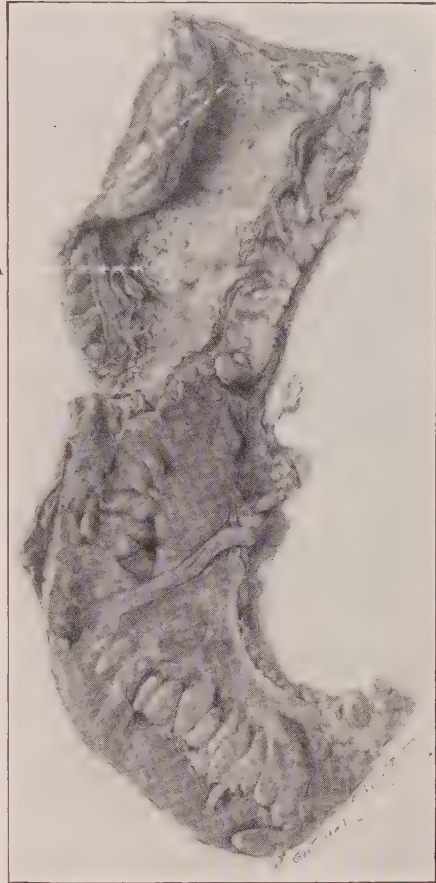


FIG. 34.—SECTION OF THE ILEUM IN A CASE OF MEMBRANOUS ILEOCOLITIS LASTING TWO MONTHS

The intestine for a distance of 2 feet was lined with a definite membrane, in which areas of ulceration can be seen (A). The upper half of the illustration shows the open ileum and the greatly thickened wall (B).



epithelium is said to contain no glands. Stricture of the intestine is rarely formed by the scar tissue.

*Lesions Outside of the Intestines.*—The mesenteric lymph-nodes are enlarged and contain bacteria. They seldom undergo suppuration. The liver is usually enlarged, and congested, and the cells are swollen and show fatty degeneration around the peripheral zone. The spleen shows at autopsy some increase in size and bacilli may be found in it.

The kidneys are swollen, soft and yellowish-gray in color. Under the capsule the blood-vessels are markedly injected. The minute changes are disintegration and marked cloudy swelling of the convoluted tubules. Some cases show well-advanced acute nephritis.

Infections of the ears may occur during the course of the disease, due to the lowered resistance. Pneumonia may be likewise coexistent but is more apt to be a terminal manifestation, in some cases only found at autopsy.

**Symptoms.**—The incubation period of this disease in childhood is the same as in adults, from three to seven days. There may be no premonitory symptoms except in some cases a temperature above the normal.

The early symptoms at once suggest a serious illness, and an intestinal disturbance. Loss of appetite and in most cases early vomiting are present.

Diarrhea is prompt in its appearance, and is usually the first symptom. The food remains more or less undigested, is soon passed, and the stools are watery and contain an excess of mucus.

The temperature is high at the onset, ranging from 103 to 105° F. Severe abdominal pain appears early, accompanied by marked straining.

As the disease advances the change in the stools grows rapidly worse. The increased frequency is of marked significance and causes much alarm. From ten to thirty movements may be passed in twenty-four hours. They eventually become small as regards the residue which is left on the diaper, but a large watery greenish or brownish stain surrounds it. The child soon loses control of the bowels. In cases clinically designated as dysentery, mucus and blood soon make up most of the residue; there is usually microscopic and in most cases macroscopic evidence of pus by the end of the first week. To the naked eye, pus and mucus are not always easily differentiated. These may be tinged with blood, or a considerable amount may be present.

In certain cases portions of membrane are passed by stool, a symptom which is indicative of marked severity.

There is little odor to the stools. It might be described as albuminous, probably from the mucus or blood, and might also be said to resemble that of wet hay. It is seldom sour, unless carbohydrates are being fed in sufficient amount to cause fermentation. Gangrenous or putrefactive odor may occur either from severe ulceration or from the character of the food.

The temperature usually remains fairly regular, but becomes lower than at the onset. The range is usually from 100 to 102° F. during the late course

of the disease. Fever is, however, characteristic of infectious diarrhea, and its persistence differentiates the disease from the acute intestinal indigestion.

Abdominal pain due to tenesmus causes the child much suffering. When the stools are almost continuous the discomfort is so marked and steady that rest and sleep are disturbed. As a result of the straining and the increasing weakness the rectum may protrude. The abdomen, at first distended, becomes flabby and sunken. Palpation usually elicits tenderness.

Vomiting is present in those cases that are manifestly toxic, or in which feeding is forced beyond the child's wishes or tolerance. In some cases it becomes uncontrollable.

The general appearance shows that the patient is steadily losing. There are many reasons for this, as the child fails to eat, the escape of water by the stools is large, the suffering and the loss of sleep interfere with rest. All of these with the toxemia and the increased oxidation of the body tissues bring the child to a state of prostration. At first the facies is anxious. Finally there is much change in the expression, the eyes becoming sunken and unresponsive, the color of the skin ashen. The abdominal wall is doughy and sunken, the tissue turgor gone. The child passes into an apathetic condition, lying relaxed and at times with half-closed eyes in a state of stupor.

The acute stage lasts for about three weeks. If large numbers of cases are considered the disease will be found on the average to have milder symptoms in older children.

**Urine.**—The reduction in frequency and quantity is the special feature of kidney excretion. Such is inevitable from the small intake and the fluid losses of the body. The urine contains a moderate amount of albumin and granular casts. Sugar is sometimes present.

Pyuria may occur at any time during the course and the urine should be examined daily under the microscope. When the temperature becomes elevated after a period of normal or uniform course, pyelitis should be suspected.

**Blood.**—In the beginning the white blood-cells are somewhat increased, usually in the neighborhood of 15,000 to 20,000. The percentage of lymphocytes is markedly decreased and the polymorphonuclear cells are high. As the disease progresses, especially in severe cases marked by prostration and toxemia, the white count may be reduced to below normal. In general one can conclude that a moderate leukocytosis or a normal white count speaks for a favorable outcome.

**Summary of Course.**—The disease begins with prompt vomiting and diarrheal stools, always containing some pus and in many cases mixed with blood. Fever is high in the first week, may remain markedly elevated, may become irregular, and in some cases, even fatal ones, stay from 100 to 101° F. The child sweats profusely, there are hoarseness, sunken eyes, prostration or collapse.

The classification as to moderate or severe has to do chiefly with the rapidity of the course and the outcome. There is much variation in the symptoms in the severe cases. While a smaller number of stools generally means a lighter case, this is not always true. Frequent stools add to a rapid tissue depletion, and are greatly to be feared. On the other hand the stools may be less excessive, and the general condition not alarming, but the toxemia continuous and exhaustion progressive. Death may occur without an exacerbation of the diarrheal symptoms. Intoxication may be sufficient to cause acidosis, in which event the outcome is unfavorable.

In the cases with moderate symptoms which recover, the blood disappears from the stools, fecal material returns, the appetite and general condition improve, the temperature gradually becomes normal, and in three weeks recovery from the intestinal symptoms may be complete.

The appearance of pieces of pseudomembrane in the stools may be of serious importance. It represents a high grade of inflammatory involvement and is usually accompanied by cerebral symptoms resembling meningitis, delirium and convulsions, high fever, vomiting, and in fatal cases, sepsis or some terminal infection, usually respiratory.

In the previously vigorous child relapses are not so probable as in the delicate infant whose nutrition has always been below normal. Primary ileocolitis with proper treatment, and without debilitating complications has a more favorable prognosis.

In the severe cases, the patient may survive only one or two weeks. It is not uncommon for the course to be protracted for a month or two. Relapses may occur. The temperature may be irregular or continuously high. The child becomes dried out, showing dryness of the tongue and mouth, sordes and some cases a marked stomatitis.

**Diagnosis.**—The clinical symptoms must remain the practical method for diagnosis. Isolation of the causative bacillus from the stools has been accomplished in some cases, but the procedure is highly technical and difficult, except by those who have had much experience in such diseases of children. The presence of so many varieties of bacteria in the stools makes the significance of any one type doubtful. Specific agglutination is not present or is incomplete in the first ten days of the disease, and may not be found at all in mild cases.

Typhoid fever in children may have constipation, or if the stools are loose, have a characteristic consistency. The movements are fluid, of the pea-soup thinness, and do not show the clear water, mucous and purulent elements so easily recognized in the gross appearance of dysenteric stools. Typhoid hemorrhage is rare and is then a late manifestation. The separation of the two diseases is difficult in many instances, particularly in infancy. One must depend upon the specific serum and blood tests of typhoid for help in doubtful cases. The spleen is less often enlarged in dysentery.

Intussusception in infancy is often overlooked, and because of the



bloody stools is frequently mistaken for ileocolitis. The acuteness of the vomiting, the suddenness of intestinal hemorrhage, the rapid appearance of the symptoms in a breast-fed infant should make the disease easily recognized. In the artificially fed infant, the chances for error are greater, as diarrhea is common and is naturally first considered. Intussusception is not accompanied, at least in the outset, by an elevated temperature, and the stool is apt to be pure blood. The white blood count is high.

Acute intestinal indigestion is the disturbance with which ileocolitis is most commonly confused. The early symptoms of each are similar and may show nothing by which one can predict the later course or outcome. The persistence of fever and diarrhea after three to five days is indicative of an inflammatory diarrhea. It is probable that some cases of acute intes-

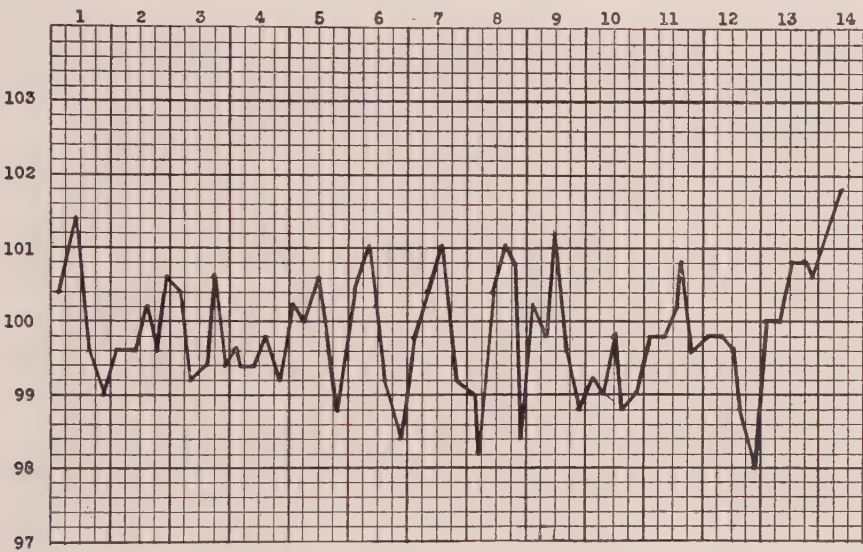


FIG. 35.—TEMPERATURE CURVE IN ILEOCOLITIS

tinal indigestion become secondarily infected with dysenteric or other organisms.

*Prognosis.*—Dysentery in childhood is always to be regarded as serious. Recovery is dependent upon many factors, such as climatic conditions, inherent resistance in the child, the virulence of the infecting organism, and proper care and nursing from the onset. Fortunately there are moderate types which recover in from one to two weeks. Others may take from three to six weeks. Occasionally the disease may last for three months.

The temperature chart above (Fig. 35) is that of a case of ileocolitis during the last two weeks of life. The symptoms had appeared mild and the daily drop in the temperature seemed to indicate that the child would soon recover. There were several factors which influenced the fatal termination. The blood chlorids were found to be greatly depleted and the child had congenital syphilis. In the last few days, stupor was constant. The essential autopsy findings were limited to a classical ileocolitis.



A fatal outcome results early in the weak infant, who seldom lives more than a few days. Deaths most commonly occur in the second week, while some may linger for several weeks.

What the outcome may be in the individual case cannot be foretold, and even in those apparently past the danger point, a cure is not assured until complete recovery takes place. Children who have continued high fever, or who in the presence of low fever show much toxemia and little reaction, have fewer chances for recovery. Continued vomiting, delirium and convulsions, and no improvement in the bloody and purulent stools, are conditions which make the outcome more unfavorable.

**Treatment.**—The dietary and nursing care of ileocolitis is the first and most important treatment. Treatment in the home is advisable only with proper conveniences and when a trained nurse is available. Otherwise, and in a family unable to undergo the nursing expense, or when there are other young children, the patient should be removed to the hospital. Proper surroundings cannot be obtained in the tenement dwelling, and the physician owes it to the child and to himself to have the advantages of a good hospital. A well-ventilated and comfortable room is necessary, and during the summer months every effort should be made to keep the child cool. The care should be individual, so that the patient will receive plenty of cool water, which is necessary, not alone for supplying the water balance, but for comfort and for the effects upon the fever, and the kidney excretion. Frequent sponge bathing and body cleanliness are necessary in diarrheal diseases, so as to avoid intertrigo and bed-sores. When daily hypodermoclysis is given, a trained assistant who is always on hand is essential.

The diet should be simple and easily digestible. *Breast milk* is the best food for the infant. Known quantities of milk which has been expressed from the wet-nurse or other donor can be given in the bottle. It is advisable to give water or weak tea alone for the first twelve hours of the illness and then begin the feeding of breast milk 1 ounce, and tea every four hours. Water should be given every half hour when the child is awake, unless there is vomiting, in which case the stomach is to be lavaged once or twice daily, and the feeding continued. Larger quantities up to 2 ounces of breast milk are to be fed as soon as the child's desire and tolerance will permit.

In the absence of breast milk for the infant, and in older children, the safest food is powdered protein (sour) milk which should be begun in one-third or one-half the usual concentration. This is attained by 1 teaspoonful of protein milk powder in each 2 ounces of water. A small amount of sugar, or a gruel, is added to the mixture.

The reaction of the stools has until recently received much consideration in the dietary treatment. As the character of the food has much to do with the acidity or alkalinity of the movements, and as both fermentation and putrefaction may coexist, it is probable that a diet well-balanced in the protein and carbohydrate elements is best for general use. Paul has sug-

gested that the urine be frequently examined for indican, and that a high amount be regarded as the indication for increasing the sugar in the food in spite of loose stools.

The concentration of protein milk and carbohydrate should be steadily increased to meet the needs of the child. This is reached when a level tablespoonful of powdered protein milk is added to each 3 ounces of water. To this amount 2 to 4 level teaspoonfuls of dextrimaltose are used.

In place of sugar a thin gruel of rice is well tolerated, in children past the age of twelve months. The cooked gruel consists of 10 per cent rice and 90 per cent water. This may be begun after the initial withdrawal of food. Protein or buttermilk may soon be added to this cereal, and sweetened with saccharin. If the child will not take sour milk, boiled skim milk may be used instead.

In children past one year of age, other articles may be added. These are meat broth, thick cereals, gelatin, cottage cheese, toast crumbs, orange juice. The loss of salts should be replaced by the feeding of vegetable juices in small amount.

The highest amount of food consistent with the safety of the individual case is indicated in such an extended disease. High caloric feeding in dysentery, based upon the experience in typhoid fever, would seem in theory to be of advantage in shortening the duration of the disease. There are difficulties in the way of its adoption. Often the child cannot be induced to eat, or is in no condition to take food. There can be no objection, however, to allowing the appetite to be the guide for increasing the amount or concentration of those foods which are suitable. Undoubtedly the use of protein or other soured milk and the increased tolerance which it gives for cereals and sugars make possible a larger dietary than was formerly used.

*Medicinal.*—At the beginning of the symptoms, castor oil is usually given. There can be no objection to an initial small dose, but no other laxative is indicated after the first day following the onset. The body fluids and the strength of the patient should be retained as fully as possible. Repeated laxatives add to the excessive peristalsis, water loss and discomfort. It is a question whether by interference with the natural restorative processes we do not do more harm than good in the use of purgatives and enemata.

The medicinal therapy of ileocolitis has not been successful, and it should be the rule to withhold drugs rather than to give them, except for occasional symptomatic reasons. If one were to criticize the most common practice in the treatment of these children, it would be that of overmedication. Polytherapy is often in evidence, with prescriptions which include bismuth, tannic acid, opium, spirits of niter, and for good measure, one or more proprietary remedies. One is not justified in giving medicine because the family expects it. The infant cannot help himself, and one's duty is primarily to the child. The family is to be informed that the treatment is almost entirely dietary and nursing.

The relief of pain, tenesmus and sleeplessness calls for occasional doses of paregoric. In inflammatory diarrhea no effect in lessening the number of stools has been observed from any safe dosage which I have used, but the child may be given temporary relief by the rest which it produces.

The best stimulant is weak tea which should be sweetened with sugar. This may be given several times daily. One or two daily subcutaneous injections of physiological salt solution, 15 c.c. ( $\frac{1}{2}$  ounce) per pound of body weight, will prove beneficial. This should be continued as long as it is readily absorbed, or while there is no evidence of edema.

It is well to keep the urine mildly alkaline throughout the disease, for its influence in preventing both acidosis and pyelitis. For this purpose sufficient bicarbonate or citrate of soda is to be given. From 10 to 15 grains of either should be administered in an ounce of water every four hours.

If highly acid or irritating stools should be present, or if the abdomen be distended with gas, the acidity may be lowered by using an absorbent. For this purpose animal charcoal has for several years found advocates in this country. My experience with it has led me to believe that the child's comfort is increased by its use.

The use of bismuth preparations or so-called intestinal antiseptics is without effect upon the diarrhea or course of the disease.

Irrigation of the bowel may be of benefit in lowering the temperature. Starch water, made by adding 10 per cent of starch to water, has frequently been used for tenesmus. If it reaches and is retained by the colon it may be of some benefit, but retention is not likely. Weak silver nitrate solutions (1 or 2 per cent) have also been employed, but it is improbable that any effect upon deep-seated lesions can be produced, while the irritating action adds to the severity of the superficial lesions. In general, one should use injections sparingly, as they are annoying to a sick and weak child. I have seen collapse caused apparently by simple enemata.

*Curative Serum.*—Treatment with specific serum has not met with much success, possibly because it has been given late in the disease. Davison, Spence, Porter and others have, however, reported some benefit with the antidyenteric serum. It does not show immediate results, but its advocates believe that it has a life-saving effect. Serum is given intravenously for four days, in doses of 20 c.c.

**Prophylaxis.**—During the entire year, but especially in the summer and fall, all water and milk for children should be boiled. When the source or purity of cow's milk is not known it is safer to use a dried milk. This has been recommended widely by pediatricians in France. Prejudice against boiled milk is founded on the change in the taste. The reduction of its vitamin C content is of little consequence since antiscorbutic foods are now given to all infants and children.

When epidemics originate in hospitals and asylums, there has been neglect of adequate precautions. Institutions should have provisions for iso-

lating cases of intestinal and other infections. The hands of the attendants, the utensils, the bedclothing, etc., should be disinfected, and the food safeguarded against contamination by human carriers or flies.

Vaccination against the Flexner type of organism is said to have been used successfully, but I have not tried it. The resulting immunity can only be temporary and is therefore applicable only in epidemics, such as in hospitals or orphanages. Wilkins and Wells were impressed by its benefits and found proof of immunity in the subsequent agglutination tests on the blood of vaccinated children. The doses of the vaccine were from two hundred and fifty million to one billion organisms. The use of any vaccine cannot take the place of proper care in the food and hygiene of children.

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## CHAPTER XVI

### THE INTESTINES (*Continued*)

#### TYPHOID FEVER IN CHILDHOOD

**Descriptive Summary.**—Infection of children with the typhoid bacillus causes less tendency to diarrhea, milder general symptoms, fewer complications and less pronounced pathological changes in the intestines. The irregular fever gives no clue to the nature of the disease—a marked difference from the characteristic and diagnostic febrile course in adults. The symptoms are markedly atypical in infancy, they may or may not resemble classical typhoid during the middle period of childhood, but from ten to fourteen years of age they are usually similar to those of adults. Each year of age shows an increasing percentage of cases, but the occurrence of typhoid in most communities is steadily decreasing. The mortality rate is low.

During the last century the exact nature of the disease has become known. In 1839, typhoid fever was differentiated from typhus fever by Gerhard of Philadelphia. Previous to that time, only a clinical separation was made, without pathological foundation. Soon afterward the method of transmission was described, and in 1880 the causative bacillus was described by Eberth, and soon found by others in the blood, urine and stools.

**Age Incidence.**—The age incidence as it has occurred in childhood is in accordance with the figures given by Percy :

2 per cent is found under two years,  
27 per cent for the years three to five,  
66 per cent for the years six to twelve.

In 429 children, Umikoff found the highest incidence between ten and twelve years, and greater in boys.

In 100 cases in children reported from an epidemic in Germany :

11 per cent were in the first two years,  
22 per cent were in the first five years,  
67 per cent occurred in school children.

The relative frequency of the disease can be best determined in extensive epidemics where the population as a whole is exposed to the source of infection. The period of infancy and early childhood has a lower incidence due to the breast feeding, and in recent years by the custom, as yet not widespread, of boiling both water and cow's milk.

In certain epidemics as much as 25 per cent of the cases occur in childhood, so that it may not be so rare as the so-called sporadic cases would indicate.

There is a marked tendency to the seasonal occurrence of typhoid. In the north temperate zone the months of August, September and October show the most cases.

**Etiology.**—The disease is spread by contamination of the drinking water through sewage or drainage, or of the water used in washing cans, bottles and household utensils, infections from the dejecta of patients or carriers, and the contamination of milk by the hands of milkers.

The examination of cooks and employees in the dairy industry is an important part of the prophylaxis of typhoid fever. In the city those who work in the milk depots are less apt to contaminate the milk, because of the abundance of water, of modern toilet facilities, and proper sterilization of bottles and containers. On the farm where the milk is produced these facilities are less available.

An interesting survey of the dairy industry in Alabama by Welch and others in the state laboratory found among the employees a rate of 3.1 per cent of carriers for typhoid and paratyphoid bacilli. Both the stools and urine should be examined. Healthy carriers may be those immune from a previous attack of typhoid or individuals who have been vaccinated and have taken contaminated food or water.

Sporadic rather than epidemic occurrences are now the rule, due to the improvement in the general water supply of communities, and the disuse of cistern and well water in most towns and cities. Open privies were long the source of danger by which the soil and water became polluted, and to which there was access by flies. It is now unheard of for typhoid to appear among children whose drinking water and milk are safeguarded. All cases that I have seen in recent years have been in children who live in small communities, or in the outskirts of the city, or who have been visiting during a vacation.

In three epidemics in Chicago the source of infection and the childhood incidence were:

1916,	Water-borne infection, 105 cases, 28.5 per cent under 14 years
1923,	Water-borne infection, 225 cases, 14.4 per cent under 14 years
1924-25,	Oyster-borne infection, 129 cases, 7.0 per cent under 14 years

In the epidemic caused by eating oysters it is interesting that one child under four years and three under nine years developed typhoid fever.

**Pathology.**—The explanation of the rarity in early childhood must lie not so much in the decreased lack of exposure as in the anatomical and immunological factors. Certainly in community epidemics, much of the population, including the children, will be exposed. It is in the young adult life that the greatest incidence of typhoid occurs.

Because of the fewer characteristic symptoms, cases in early childhood are sometimes overlooked. The disease would be more readily recognized if we remembered that it resembles a septicemia with little clinical evidence of pathological disturbances in the intestinal tract. The microorganism enters by way of the digestive tract but does not remain localized to it. The bacillus finds a lodgment in the mucosa and soon afterwards reaches the wall of the ileum from which it invades the blood stream. The general condition is therefore one of septicemia.

A mild inflammatory reaction is produced by the bacilli and their toxins. The lesion is made up of accumulations and proliferation of endothelial leukocytes which are capable of digesting blood-cells and of migrating to lymphoid tissue. As the toxins are absorbed from the lesions in the intestinal wall, changes develop in the adjacent lymphatic vessels and nodes. The lymphatic vessels contain accumulations of these leukocytes, as do also the mesenteric lymph glands. The characteristic lesions are in the lymphoid tissues of the ileum, where Peyer's patches and the solitary lymph follicles show an inflammatory reaction resulting in a thickening of the inner coats of the intestine. In severe cases, especially in older children and adults, the accumulation of phagocytic leukocytes causes a blocking of the small blood-vessels (thrombosis). Necrosis and sloughing may result in hemorrhage, and if deep enough, in perforation. No scar tissue develops from the local lesions in the intestines.

Peritonitis can exist without perforation probably due to direct extension from the lesion in the intestinal wall. In fifty cases of peritonitis without perforation, reported by Greenwald, two were in children, neither of whom was above two years of age. A serofibrinous peritoneal exudate was found in an infant of sixteen months, following the drainage of which recovery took place.

The increase in the size of the spleen is due to the marked accumulation of endothelial leukocytes. The same reactionary mechanism affects the liver and may cause changes in the heart, lungs, bone marrow, kidneys, suprarenals, pancreas and testes. The gall-bladder and the urinary bladder may harbor bacilli for months, but so far as is known without causing local lesions therein, except in adults where persistent infection of the biliary tract may favor the development of gall-stones.

Abscesses of the muscles, ribs and other long bones are due either to mixed infection or directly to the typhoid bacillus. I have known of one case of osteomyelitis of the ribs from the discharge of which a pure culture of typhoid bacilli was obtained months after the intestinal disease had disappeared.

**Symptoms.**—The symptoms in infancy and early childhood are less characteristic and because of the infrequency of the disease are apt to be mistaken for those of some other infection. Cases at any time during childhood, however, are apt to present features which are markedly different

from the adult course. The period of incubation averages from ten to fourteen days, but may show the same tendency to vary found in later life.

During infancy the disease begins with sudden high fever, stupor and vomiting. The temperature remains high and is therefore described as of the "plateau" type. Diarrhea is usually present from the start. There may be signs of meningeal irritation with twitching, convulsions or stiff neck. Cough at this time of life is a rare symptom. There is less tendency to the development of a rash. The tongue is white, with a red, triangular-shaped area at the tip. The course is shorter and less intense, and the prognosis good.

Following infancy the onset in early childhood shows the same tendency to a sudden and marked rise of temperature. This early appearance of high fever and of definite daily remissions is entirely different from the classical "stepladder" febrile onset in later life. During the course of the disease there is a wide excursion from 105° F. in the later half of the day to a low point of only one or two degrees' elevation in the early morning. The fever is irregular throughout, and lasts in the majority of cases less than twenty-five days with a considerable number under fourteen days. As short a duration as one week has been reported, but this is rare and in such a short course the true nature of the disease is apt to be unrecognized. According to Griffith one-fifth of the cases in children may have a persistence of fever for more than twenty-one days. The decline of the fever by lysis is the typical course, but there may be a critical drop occurring in two days.

Stupor is to be expected in the first weeks of typhoid, and exceptionally, initial shivering and convulsions. Nosebleed at the onset occurs in older children with the same frequency as in adults, but is less common in the young.

Vomiting occurs early and it may be a constant symptom but is in no way diagnostic. It is of toxic origin, the stomach showing no pathological changes.

The appetite is not normal, but is relatively less disturbed than in adults. The child if left to its own inclinations would not eat and would take little except cold drinks. The child may be induced to take considerable food during the course of the illness, much more than was thought possible or advisable during the days when the starvation treatment was practiced.

From the foregoing it will be seen that in the beginning the child presents a picture of a septicemia, the cause of which is not readily suggested by any constant or diagnostic signs. As the disease extends beyond the second week, the symptoms become more characteristic. As soon as typhoid infection is suspected, it is common to examine the blood. It is usually stated that culture of the blood will be positive during the first week of typhoid, and the Widal in the second or later weeks. The physician will be disappointed if he expects early or constant help from these tests in chil-



dren. Blood cultures are rarely positive in the first week and in not over half of the cases at any time. The Widal agglutination may not become positive until late in the course, and in certain cases not at all, at least during the time that the child is under observation.

The stools are not so uniformly diarrheal as in adults. Probably one-fourth of the cases have constipation and the remaining three-fourths may have either normal stools or diarrhea. The condition of the stools is apt to vary during the course. Constipation may give way to diarrhea in the second or third week. When diarrhea occurs the stools are not so frequent nor so dehydrating as in dysentery. They are characteristic of typhoid, being of the "pea-soup" consistency and color and with an offensive odor. They are thin enough to pass unassisted through a medium-sized catheter. Diarrhea is found more often in the severe cases.

Abdominal distention is not a feature in childhood and appears late if at all. A few cases have a sunken abdomen. Meteorism may be absent entirely or appear late in the disease. Gurgling in the lower abdomen is more constant than in adults. Abdominal pain is said to be present in about 25 per cent of cases, when it will be found in the ileocecal region.

The physician constantly watches for enlargement of the spleen for help in the diagnosis. The illness may last several weeks before the spleen becomes demonstrable by palpation. At some time during the course it becomes enlarged, in 87 per cent of cases. Its size may vary from that at which it may barely be recognized under the left costal border to that of a definite mass felt one or two inches below.

The *rose spots* are frequently gone by the time the child enters the hospital, as the rash leaves quickly. It is present in 50 per cent of cases. The lesions are few and may be overlooked, and occur mostly in the first weeks, at times not until the spleen is enlarged. They are not elevated, are pale red, and fade out on pressure by the finger, to slowly reappear.

*Bronchitis* is so constant as to be regarded a symptom of typhoid fever in childhood. Where it appears early it adds to the confusion in the diagnosis. Coarse bronchial râles and a cough are present. Pneumonia should be considered as a complication.

*Nervous symptoms* are found at some time during the disease, more commonly in older children. There is seldom the characteristic typhoid mental state, but rather excitability, and occasionally delirium or convulsions. Typhoid meningitis is mentioned in the literature and in one case coexisted with tuberculous meningitis. Four cases of purulent meningitis with typhoid bacilli in the spinal fluid have been collected in recent years. In typhoid fever the development of meningitis may be unsuspected if a delirium has preëxisted.

*Urine.*—The quantity is reduced, due to the fever, and to diarrhea. In cases where there is small intake of fluids the reduction in urine is still

more marked. There is usually some albumin present and a few casts. Typhoid bacilli can be demonstrated in certain cases.

*Blood.*—The characteristic finding in the blood is the leukopenia, the white count being reduced below 10,000 in older children, but less affected in younger children. Large mononuclear lymphocytes are increased in percentage. There is usually found a diminution in the hemoglobin as the disease progresses. The red cells are seldom below 5,000,000. The blood-pressure is only slightly diminished during the disease. It is well to determine the pressure in the individual case, so that the subsequent pressure reading may be therewith compared when perforation is suspected.

The pulse is relatively slower than the fever would indicate, except in complicated cases. As the fever subsides the pulse goes quickly to a still lower level, in some cases 50 per minute.

During the progress of the disease a systolic murmur is common, due to myocardial degeneration resulting from the continued toxemia, but it disappears during convalescence.

The following is a report of a case which illustrates the difficulty of a typhoid diagnosis in childhood, because of irregular features:

A five-year-old boy had become sick with a sudden high temperature, rapid loss of weight, nervousness and irritability. The stools during the first week of illness had been greenish and liquid, but soon became constipated. After three weeks of fever, during which he was not bedfast, he was brought to the hospital. The appetite was poor, the intestines were markedly distended, but there were no other symptoms referable to the digestive tract.

The routine examination disclosed nothing which was positive. The temperature varied from 101 to 105° F., never reaching normal. Tuberculosis, pneumonia, otitis media, pyelitis and malaria were ruled out. Cultures of the blood, urine and stools and agglutination tests were repeatedly negative. In the fifth week of illness the spleen became palpable, the Widal test positive, and the stools diarrheal. The white blood count was usually around 10,000 cells, and instead of the normal lymphocyte percentage there was a definitely larger number of polymorphonuclear cells ranging from 55 to 69 per cent. The fever decreased by lysis and no complications occurred in the seven weeks of illness.

The frequency of the principal symptoms is shown in the following percentages obtained from one hundred cases in children (Bischoff):

	<i>Per Cent</i>
Positive agglutination blood test, Widal (1:400 the usual titer).....	88.0
Diazo reaction in urine.....	73.4
Bacteria recovered from the blood.....	71.8
Leukopenia .....	71.0
Rose spots.....	63.0
Diarrhea .....	52.0
Enlarged spleen.....	44.0
Typical fever.....	17.0
Bacilli in stools.....	13.0
Bacilli in urine.....	2.3

**Course.**—The duration is variable, the minimum reported in childhood being from ten to fourteen days. It is more correct to say that light cases have a duration not exceeding twenty-five days. Moderate cases have mild complications and average thirty-six days. Severe cases are not to be judged alone from the height of the fever but from the protracted course of it, and from severe complications. Duration of three months has been reported in cases with relapses and other complications.

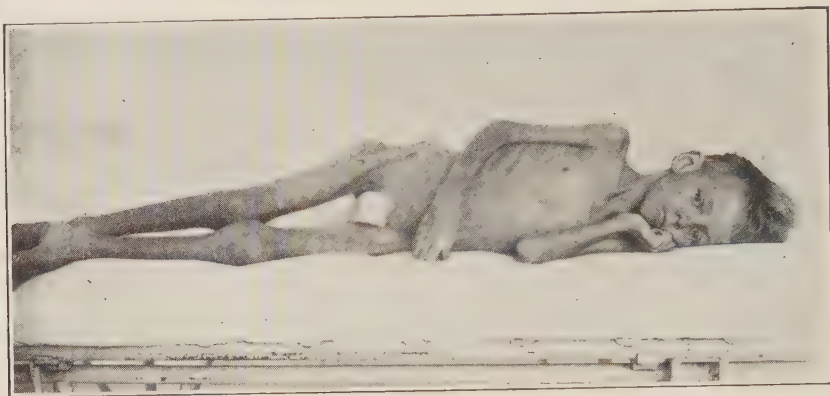


FIG. 36.—EXTREME EMACIATION IN PROTRACTED CASE OF TYPHOID FEVER

**Relapse.**—This occurs in about 8 per cent of cases. It is now generally considered that the chief cause of relapse is starvation of the body tissues, possibly due to insufficient ingestion of food. Opinions have varied as to the influence of such causes as leaving the bed too early, chilling, severity or brevity of the attack, and so-called typhoid poisoning. If relapse occurs, it is inherently due to the delay in the development of immunity. Cases relapse in varying periods from one to twenty-one days after the temperature has reached normal. The relapse is shorter and milder than the original attack. Recurrent fever is found in about 4 per cent of cases.

**Complications.**—Light cases have lower fever and no complications or relapses. Complications are usually evidence of the severity of the disease.

The most frequent complication in children is pneumonia, due to other organisms than the *B. typhosus*. It is a result of the weakened state of the individual. The outcome is unfavorable.

Hemorrhage from the intestine is occasionally seen. It is indicative of ulceration, but does not necessarily go on to perforation. Hemorrhage is a disquieting symptom, but seldom serious. It is present in about 3 per cent of cases.

Perforation of the intestine is rare. It is indicated by acute pain below the umbilicus, and the appearance of peritonitis with rigidity of the abdominal wall. The symptoms are those of shock with a drop in temperature, blood-pressure, and an increase in the pulse rate and respiration.

Acute deafness and otitis media, furunculosis and other suppurations

are not infrequent. Typhoid osteomyelitis of the ribs and of the other long bones is occasionally reported. Pyelitis from typhoid or colon bacilli sometimes occurs during the disease in childhood. Gall-bladder infection with a persistence of the typhoid carrier state seldom results in childhood.

The thinning of the hair is of frequent occurrence during or following the long febrile course in severe cases.

Aphasia was reported in twenty-one cases by Morse, some of these due to embolism.

**Diagnosis.**—It is considered that the blood of the patient will usually yield a positive culture of *B. typhosus* during the first week of the disease, and that the agglutinating phenomenon (Widal test) will be present in the second and later weeks. If this were always true the diagnosis would be greatly simplified. But there are so many exceptions in practice that either repeated tests must be made or the diagnosis must be arrived at by clinical evidence alone. In epidemics and in typical cases the laboratory confirmation is not usually necessary, but in the isolated cases, and in the young child, an extensive experience in the symptomatology of typhoid at this time of life is needed to make it possible to recognize the disease without laboratory assistance.

Such symptoms as lack of appetite, malaise, shivering, diarrhea and nosebleed are not constant, but when present are of help in suggesting typhoid infections.

Ehrlich's diazo reaction appears in the urine in the majority of cases among children. The reaction becomes weaker as the condition improves and more marked in severe cases and at the onset of unfavorable developments such as relapse.

A simple test, De Silvestri's, can be made with the typhoid urine and is one of easy application (see Appendix).

**Differentiation.**—It is probable that paratyphoid infection is present in the child who has symptoms resembling typhoid but in whom the typhoid bacillus agglutination and the blood cultures have been repeatedly negative throughout.

**Pneumonia.**—In earlier practice the designation of "typhoid-pneumonia" was common, owing to the lateness of diagnostic signs of the typhoid; or to the mistake in assuming that the cough and râles of an associated bronchitis were pneumonia; or to the fact that pneumonia frequently presents delirium and an abdominal distention. Pneumonia is a common complication of typhoid, but is due to pneumococcus or other respiratory organisms. The high leukocyte count in pneumonia will differentiate it from uncomplicated typhoid, as well as the pneumonic physical signs and the typical shadows in the roentgen ray picture.

**Miliary Tuberculosis.**—Abdominal tuberculosis or generalized tuberculosis of the lungs and meninges present symptoms which are easily confused with those of typhoid. The constant fever, loss of appetite and weight,



splenic enlargement, abdominal distention, and cerebral irritability are often present both in tuberculosis and typhoid of childhood. A positive tuberculin reaction (intradermal), and a radiogram which is positive for a generalized miliary disease of the lungs will assist in the diagnosis of tuberculosis.

*Pyelitis.*—One of the commonly overlooked diseases mistaken for typhoid fever is pyelitis. Almost all the physical symptoms of typhoid may be present in pyelitis. There is leukocytosis in pyelitis and much pus in the urine, and a negative Widal reaction.

Toxemias are of common occurrence in childhood, and they may resemble typhoid fever. In addition to pyelitis, chronic or latent mastoiditis, sinusitis, infection of the teeth and of the cervical and bronchial glands may produce a protracted febrile course which is puzzling and suggestive of typhoid.

*Dysentery.*—This most commonly enters into the question of diagnosis. The temperature does not go so high, the stools contain much mucus, pus and blood. They are entirely different from those of typhoid, which in children are often constipated or of the pea-soup consistency.

**Treatment.**—*General.*—The general nursing care is the most important part in the treatment of typhoid, and deserves first consideration. Much can be done for the comfort and welfare of the child by the observance of cleanliness and body hygiene. The mouth and teeth should be kept clean. Sponge baths, if well borne, should be given several times daily. Rubbing with alcohol should follow the bath. Bed-sores are to be prevented. The bed linen should be kept fresh. Involuntary stools are to be anticipated by the wearing of a thick diaper.

Much water is needed by the child because of the high temperature and the small food intake. There is no objection to giving orangeade, lemonade or weak tea.

The nursing<sup>1</sup> should be in charge of a graduate who will chart not only

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<sup>1</sup> An excellent routine for nursing care has been suggested by Dr. Julius Hess as follows:

1. Temperature, pulse and respiration every four hours
2. Diet as directed
3. Sponge or tub bath when the temperature is above 102.5° mouth, or 103° rectal
4. Colonic flushing with normal salt solution every morning
5. Hemorrhage or perforation is indicated by:
  1. Collapse: pallor, rapid pulse and perspiration
  2. Discharge of blood from bowels
  3. Sudden fall of temperature
  4. Sudden pain in abdomen and abdominal distress
  5. Vomiting
6. Treatment of hemorrhage or perforation:
  1. Notify doctor immediately
  2. Stop all diet and orders
  3. Elevate foot of bed.
  4. Apply ice coil to abdomen
  5. Keep patient at absolute rest

Chlorinated soda for disinfecting excreta

Formalin for linens

the usual temperature and bedside notes, but who will record the amount and value of the food taken. She will be of much help in getting the child to eat a sufficient amount, as well as in the general nursing care. The recognition of untoward symptoms can be made more promptly by the experienced observer who is present at the bedside. Severe cases are better handled in the hospital.

*Medicinal Treatment.*—Temperatures under  $104^{\circ}$  F. seldom need antipyretic drugs. When hyperpyrexia occurs with extreme restlessness the occasional use of acetyl salicylic acid (aspirin) has proved safe and of prompt action. In two recent cases in children under six years of age the temperature dropped from  $105^{\circ}$  F. to near normal each time that this drug was used and remained there for several hours.

Severe diarrhea with marked water loss is benefited by the effect of opium upon the peristalsis. Paregoric is convenient of administration, always obtainable, and of benefit for this purpose. The dosage should be based upon the age of the child, and sufficient used to bring results.

The custom of giving calomel in typhoid as well as other diarrheas is useless if not injurious. As much intestinal quiet as possible is needed so that hemorrhage and perforation are not favored.

Urotropin (hexamethylenamin) is sometimes given for the bacteriuria of typhoid. The dosage should be large enough to render the urine sterile, and combined with acid sodium phosphate in case the urine is not already acid. One grain of each drug for each year of age may be safely given, at eight-hour intervals.

There is no specific drug in typhoid. The custom is all too prevalent of giving too many medicines in childhood. In typhoid, where the maintenance of an adequate diet and body weight is of chief importance, the use of much medicine will upset the stomach and disturb the child.

*Dietary Treatment.*—In recent years the high caloric diet for typhoid fever has come into more general use. In severe cases fluid may still be the only form of nourishment which the child will swallow, but as soon as semisolids are taken it is well to use them. Cream, milk, eggs, gruels, gelatin, fruit juices, apple sauce, sugar and bread crumbs will enable the dietary to contain sufficient calories for body maintenance. Effort should be made to secure a food intake of at least 32 calories per pound of body weight. However, the appetite is the best guide to the amount and kind of food.

The essentials in treatment are the cultivation of the appetite, the giving of sufficient food, the maintenance of body cleanliness, and the prevention of transmission of the disease to others.

*Surgical Treatment.*—Perforation of the intestine calls for immediate operative intervention. Laparotomy, with repair of the perforated gut and drainage, is essential. The results of prompt surgical attention are often striking.

*Typhoid Vaccine in the Therapy.*—In the period when Russell and

Wright were reporting their results with vaccine therapy, I used it in all cases, of which the number was considerably larger than in recent years. It was found of no benefit or use in the case of moderate severity and short duration. Vaccine treatment did not help the seventy-seven cases treated by Morquio. In long-continued cases, however, with delay in the appearance of a positive Widal reaction, it is probable that convalescence is quickened by large dosage in weekly intervals, as suggested by Knox.

*Prophylactic Treatment.*—Vaccination for the prevention of typhoid should be used at three-year intervals in children beyond the age of five years, especially in communities where typhoid is epidemic, and for children who are to go to the country or resorts for a vacation. It is the custom in my practice to suggest preventive measures, and many parents agree to its use. The doses are given at seven-day intervals, usually 100,000,000 the first week, 500,000,000 the second, and 1,000,000,000 the third. No unfavorable reaction occurs, some children having a sore arm for a few days after the injections. I have seen typhoid occur in three years after the vaccination; so that the repetition of the injections is to be recommended each three years when exposure is possible.

The occurrence of a case of typhoid should cause an immediate search for the source of contamination, whether in the water, milk, other food or from a carrier. The water supply in each community is made safe by filtration. Where such precautions are not taken the water should be boiled.

The urine and stools from the sick room should be disinfected in a 5 per cent carbolic acid solution. Dishes and bedclothes should be boiled for half an hour.

**Prognosis.**—The disease is self-limited. One attack renders the individual immune permanently, and the blood continues to have a certain degree of agglutinating power for the *B. typhosus*. Children regain their weight rapidly after the fever, and the other evidences of convalescence quickly develop.

The mortality is increased by complications of pneumonia and suppurative diseases, by severe symptoms with long-continued high temperature, by hemorrhages, intestinal perforation and peritonitis. It is difficult to state the percentage of deaths in typhoid fever in children because of the wide variance in reported figures. In the series quoted by Unihoff the mortality is 2.5 per cent, and this is the death incidence in Knox's forty-five cases from two to twelve years of age. In fifteen hundred cases reported by Morquio in Uruguay, the mortality was 9 per cent.

As a rule the younger the child the milder the course. In general the prognosis is favorable due to the lightness of the disease and the small amount of pathological changes in the intestines.

The mortality decreases when children are fed sufficient nourishing thick food and plenty of liquids. *Mixed diets* improve the general condition, the appetite and the morale of the patients. The temperature is lower,

the duration shorter, and the weight does not become so low. Intestinal symptoms are milder and the stools better. Complications of hemorrhage, perforation and relapses are fewer, as are also furunculosis, edema, otitis media, bed-sores, and pneumonia.

## PARATYPHOID FEVER

**General Consideration.**—Paratyphoid fever is of occasional occurrence in childhood, but is still rarer than typhoid. It is usual for the disease to be diagnosed as typhoid except in infancy when it is ordinarily regarded as dysentery. Probably many atypical cases of typhoid failing to give a positive blood culture or a typhoid agglutination test are due to paratyphoid infection. Because of its highly contagious nature, its early recognition is important in order to prevent an epidemic. Numerous clinical types have been described, based on the resemblance of their symptoms to other diseases, and are designated as gastro-enteric, choleric, malarial, septicemic or influenzal. The outbreak of well-described endemics in Germany in recent years suggests that diarrhea due to paratyphoid colitis, as it is there called, is more common than heretofore suspected. The diagnosis needs the correlation of the clinical, bacteriological (stool), serological (Widal) and epidemic findings.

**Etiology.**—*Incidence.*—In the United States few cases have been reported in the first year of life. The group of organisms of the typhoid-dysentery type, known as Morgan bacilli, are peculiar to the diarrheal stools of infancy. All of the three groups of this Gram-negative bacillus were found in cultures from such cases in a New York State epidemic. One is warranted in depending upon the reports of a careful laboratory as to the presence of a positive culture in the stools, and the specific nature of the Widal test.

Numerous writers abroad have reported small epidemics. Individual cases have been proven bacteriologically. Occasional epidemics are reported which include children of all ages. The disease should be considered as a possibility in every epidemic of gastro-enteritis and the stools should be examined.

**Bacteriology.**—The paratyphoid microorganisms belong to the colon-typhoid-dysentery groups, and are classified as A, B, C, and D, according to their cultural characteristics and their power of causing clumping in known cultures. *Paratyphosum A* produces a milder type of disease, and *B* more severe. The disease may be contracted from contamination of food, especially milk, by the hands of a carrier. One case occurred in a hospital child who ate a piece of candy given by a convalescent paratyphoid patient. When a case develops in a child who has not been out of the hospital or away from home for weeks, the source of the transmission is probably through some near-at-hand carrier or in the food contamination. Serum



agglutination is regarded positive in dilutions of 1:50, 1:100, 1:200 or 1:400.

In the feces, bacilli are recovered from the second to the sixth day. The urine shows no typical bacteria, but colon and other atypical bacilli of the paratyphoid group are found.

**Lesions.**—Clinically, in the severe cases, the morbid condition consists of persistent fever, prostration, tenacious diarrhea and the rash. In infancy the tissue change is principally a catarrhal inflammation of the mucosa of the ileum and entire large intestine. At any age there is swelling and infiltration of the lymphatic tissue of this portion of the intestine. The blood-vessels of the mucous membrane are dilated. Some hemorrhage is usually present, but ulceration is even rarer than in typhoid fever of childhood. Cloudy swelling due to toxic degeneration has been reported as present in the liver, kidneys and heart. Autopsy reports are few. In one case at autopsy the heart was found soft and flabby with evidence that death had been caused by myocardial failure. The mucosa of the ileocecal regions showed hemorrhagic points and swollen Peyer's follicles. The constant presence of mucous and hemorrhagic stools indicates that the disease must be regarded as a catarrhal colitis. These cases do not have the straining and tenesmus found in dysentery, so that it is probable that the deeper layers of the intestinal wall are not involved.

**Symptoms.**—The types of this infection are based largely upon the variation in clinical severity and the predominance of certain symptoms. With infants it resembles gastro-intestinal intoxication and mild enteritis, while in older children it more nearly coincides with the typhoid septicemia of childhood.

*In Infancy.*—The gastro-intestinal type is the one usually found in infants. It resembles an acute gastro-enteritis with vomiting, fever, abdominal pain, greenish stools containing mucus and blood. The course is much shorter than dysentery, usually from two to three weeks. In young children, cases may be so slight that paratyphoid infection may be unsuspected. The loss of weight is a marked symptom because of the rapidity of its development. The fever in infancy resembles that of intestinal intoxication but remains high for only one or two days, after which it gradually comes down to normal. At this time of life the blood-picture is usually one of leukocytosis, averaging about 16,000 cells to the cubic millimeter, chiefly lymphocytes. Seldom can a blood culture be obtained, the Widal reaction is most common at the end of the first or the beginning of the second week.

The general condition is ordinarily good. The striking feature of the stools is the blood which is evident in most cases, and can be found with the ordinary tests.

*In Older Children.*—The typhoid form is the type usually presented by older children. The typhoid form is so named because of its resemblance to the clinical symptoms of typhoid fever and its tendency to stupor. Some

children are not bedfast and the symptoms are mild. After an incubation period of from one to seven days, symptoms of the disease appear either gradually or abruptly. The fever is usually highest at the onset of the disease, and an initial symptom of shivering or a convulsion may occur in younger children.

Much importance has been placed by Stolkind upon the eruptions of paratyphoid fever which differ considerably from true typhoid. Herpes may develop around the mouth, the tongue is coated and a sweat rash may cover the body. The marked perspiration and sweat rash persist throughout the course—a characteristic of this disease rather than of typhoid. Rose spots are not so common. They sometimes appear in the first or second week of the disease and may be limited to a few lesions or distributed generally over the body. Rashes resembling urticaria, scarlet fever or measles have been reported. It is probable that mixed infection may occur, and be responsible not only for the different types of eruptions, but for other variations in this disease.

In older children subjective symptoms will be headache and pain throughout the body, especially in the abdomen. There is shivering due to chilliness. The temperature is not so high as in typhoid and the fever runs a shorter course. The pulse is similarly slow. The spleen does not always become enlarged. The duration of the disease is from two to three weeks, but may last as many as four, followed by a favorable outcome. Relapses may occur.

The stools vary much in older children. In the mild cases they may resemble a simple diarrhea, but in others the stools may be liquid or pea-soup, copious, yellow or may contain blood. In other cases there is no diarrhea, or the early looseness may be followed by constipation.

In certain cases the resemblance to influenza has been noticed. At the onset the throat may be red; the tongue is always coated. Throughout the case reports one is struck by the not infrequent mention of prostration which may be pronounced. The heart may fail with symptoms of cyanosis of the face and extremities, dyspnea, fast pulse, subnormal temperature and collapse, though this outcome is rare.

In recent years, it seems to have become well established that contamination of food resulting in poisoning is due to the presence of paratyphoid or associated organisms. The initial intoxication in food poisoning is much the same as that just described as occurring with paratyphoid fever, and is accompanied or followed by diarrhea and marked weakness. Rapidly fatal cases are reported resembling cholera infantum. The urine may be suppressed; the skin shows marked redness. The nervous symptoms are stupor and a tendency to convulsions.

**Complications.**—A rare complication of paratyphoid infection is the involvement of the meninges. The bacillus may be recovered from the

spinal fluid, which is cloudy, due to the large number of leukocytes, chiefly of the polymorphonuclear type. In such a case death is inevitable.

Suppurative arthritis has been reported. Perforation followed by abscess or general peritonitis may occur. The disease may cause a latent tuberculosis to become manifest. As a rule there are no unfavorable sequelæ.

**Diagnosis.**—The disease is to be regarded as epidemic, characterized by mucous and somewhat bloody diarrhea, and, as a secondary manifestation in infants and young children, by a mild form of intestinal intoxication. The occurrence of diarrhea during the fall and winter months especially should suggest an infection by the paratyphoid organisms. Colitis from this cause is more frequent than reports in this country would indicate, and will be more commonly recognized when stools are cultured and the Widal test for paratyphoid agglutination employed routinely.

**Prognosis and Treatment.**—The handling of these cases is similar to the care needed for typhoid fever, and consists of dietary measures. Infants should have tea or protein milk during the first day or two, after which sugar may be added in increasing amounts. When dehydration occurs the subcutaneous injection of salt solution is advisable. Within one or two weeks from the beginning, the number of stools becomes reduced, the character more formed, and blood disappears. Fever seldom lasts longer than a week in infants, or two weeks in older children. The weight is rapidly regained. The urine soon becomes normal.

Acute gastro-intestinal disturbance from food contamination due to the paratyphoid organisms should be treated at once by lavage and laxatives. The threatened collapse and prostration are benefited by external heat and subcutaneous injections of salt solution.

Prophylaxis is possible by the use of paratyphoid vaccine, but as yet its use is impractical in young children. In older individuals who are given typhoid vaccine, it is well to include the paratyphoid strains. Stools, urine and all articles contaminated by a known case should be treated antiseptically.

## CHRONIC ULCERATIVE COLITIS

**Descriptive Summary.**—A form of chronic ulcerative colitis known in adults has recently been found to occur in childhood beginning after the age of eight years. The characteristics of the disease are (1) severe ulcerative lesions which gradually involve the entire large intestine, (2) frequent hemorrhagic and purulent stools, (3) long course of the disease, (4) marked emaciation, (5) and the lack of response to the usual medical treatment. Surgery is to be recommended.

**Etiology.**—Comparatively little has been written about this type of chronic colitis in children. The disease is entirely different from that

common form of dysentery which is frequent in childhood and which runs an acute or subacute course. The writer has seen three cases, two of which are included in the four reported in 1923 by Helmholz. No microörganism has as yet been identified, although Bagen believes that a diplococcus is the cause. Undoubtedly cases of this disease have been overlooked and have not been differentiated from ordinary dysentery. Amebic and tuberculous diarrhea and cases of typhoid with hemorrhagic stools might well be mistaken for chronic ulcerative colitis, because of the similarity of the clinical manifestations. The etiology of these other types of intestinal infections is known and it is usually possible to find the causative organism. The stools and blood are never found to contain any constant microörganism which suggested a causative relationship. Whatever the type of microörganism that is present in the beginning of the disease, the infection becomes mixed as the course progresses.

**Lesions.**—In the beginning of the disease, the local process in the bowel consists of ulcers low in the rectum. This is characteristic. The lesions gradually extend to the sigmoid and finally to the entire colon. The mucous membrane of the intestine becomes inflamed, ulcers form and the wall of the gut becomes thickened. The mucous surface presents a glazed appearance. This appearance corresponds with the slimy glistening growth on Endo's culture medium, an examination of which shows Gram-negative rods, doubtless belonging to the colon group. The ulcers vary from small size to widespread involvement of the mucosa with granular denudation. Much hemorrhage occurs, consisting of fresh blood and clots. In the radiogram there is seen marked narrowing of the lumen of the gut. There is no question in this disease about the disappearance of the normal sacculations (haustra, tucks or pouches caused by the longitudinal bands which are shorter than the colon itself). This is a characteristic of the disease. Kennedy states that the sacculations reappear after recovery.

From the beginning of the ulcerations in the rectum, the process extends upward and finally involves the entire large intestine. In one of my cases the rectum became perforated and the perirectal region a large abscess cavity.

The pathological anatomy at autopsy shows irregular ulcerations of various sizes in the mucous membrane with very little fibrinous exudate upon the ulcers. The edges may be undermined. The ulcerative process may extend through the muscular coat as far as the peritoneum. In some regions there may be scars of healed lesions. The mucous membrane between the ulcerated portions is thickened, congested and edematous. The process may extend to the ileum but is usually confined to the cecum, colon, sigmoid and rectum.

**Symptoms.**—The history of emaciation accompanying a chronic bloody diarrhea gives the clue to the diagnosis.

The disease begins acutely with diarrhea of a bloody mucous character.



Tenesmus gradually appears. The appetite is always poor. The loss of blood is considerable. This with the rapid evacuation of incompletely digested food produces a marked loss of weight. The temperature is little elevated except when a complication occurs. The picture of the child is pitiable and the suffering is intense.

Examination by the proctoscope reveals ulcers in the rectum and later in the sigmoid portion of the colon. All chronic cases should receive an anesthetic and be given a sigmoidoscopic examination. The radiogram is of value when the involvement of the colon is extensive. Deep ulcerations into the muscular coat probably obliterate the normal sacculations and the absence of these is noted by the roentgenologist. The colon is much narrowed in diameter. Evans has reported severe cases in which the large intestine is a veritable abscess or bag of pus.

The long course of the disease easily separates it from dysentery. The usual duration is for many months, and in some cases for years.

The following synopses of my three case histories will describe different features of the disease:

1. Unusually severe ulceration with marked sloughing, operation (cecostomy) and death.

George B., eight years old, had scarlet fever one year before, during and following which he passed blood in the stools. Tonsillectomy apparently caused a marked improvement in the boy's condition with a subsidence of the hemorrhage. But within a few months he developed bloody purulent diarrhea, and high fever. He was brought into the hospital because of the severe ulcerations, fissures and sinuses in the anus and lower rectum. Sloughing of the sphincter with incontinence resulted. He soon became bedfast and the muscles wasted and flabby. The fever and the number of stools varied markedly. There were periods of remission when the boy seemed to be about well. The appetite, however, never became normal.

The perirectal abscess caused much destruction of tissue. Ulcerative stomatitis added to the boy's suffering. There was no change in the white blood count, but the red cells and hemoglobin showed a secondary anemia. The stools were negative for ameba, tubercle and dysentery bacilli. Local treatment by injections and by curettement of ulcers was at first beneficial, but a relapse soon occurred.

The child was then taken to the Mayo Clinic where he was operated upon by Dr. Balfour. A cecostomy and appendectomy were performed. The boy's emaciated state, however, made him a poor operative risk and he died soon afterward from respiratory infection.

Autopsy showed an ulcerative inflammation of the entire large bowel.

2. Markedly hemorrhagic stools, emaciation, operation (ileostomy) incomplete recovery.

Hazel L., aged eleven years, had always been thin and much below weight. Dysentery began four months before with flakes of blood in the feces but no pain. At that time the blood was seen to come from a fissure. She then had a light form of bronchopneumonia for two weeks and the bloody mucous stools began to be from ten to twelve daily. A loss in weight was soon evident.

When the child came into the hospital it seemed probable that the dysentery

was amebic. The stools were fluid, reddish yellow, acid, and contained mucus. Microscopically no parasites, ameba or suspicious bacteria were found. Her physical examination showed severe emaciation, pallor of the skin and mucous membrane (the hemoglobin was 65 per cent), the child too weak to walk, the blood contained 19,000 white cells, 2,500,000 red cells, polymorphonuclear leukocytes 74 per cent, which is an abnormal increase in childhood; these cells were poor in granules; the blood-platelets were greatly increased, there were no blood parasites nor microorganisms. The urine contained no blood nor any other abnormality. There was a daily temperature range of from normal to several degrees above. A blood transfusion of 800 c.c. was of temporary help.

At operation which became necessary from ineffectual medical treatment, it was found that her ulcerative lesions extended all the way to the ileocecal valve. An ileostomy was the type of operation performed by Dr. Sistrunk. Helmholtz could find no ameba or dysentery bacilli in this case.

The child made a slow but steady improvement and at last reports had not recovered entirely but had gained in weight and made a general improvement.

3. Bloody and purulent diarrhea, marked emaciation, endocarditis, infected tonsils, operation (ileostomy), complete recovery.

Troy S., an eleven-year-old boy, for five months had been having a gradually increasing diarrhea. This had become bloody and purulent with very frequent stools. Emaciation became extreme. A mitral systolic murmur had developed during the course of the disease and considerable cardiac dilatation was present. The boy had been bedfast for many weeks and had developed contractures of the knees. His body weight was 52 pounds (about a seven-year-old average). Secondary anemia had developed; hemoglobin 55 per cent, red cells 3,000,000, while cells 7,000, polymorphonuclears 53 per cent, small lymphocytes 33. The stools were soft and mushy with much pus, mucus and blood, no fat nor parasites. The diagnosis of chronic ulcerative colitis was definite and the associated endocarditis suggested that the cause of both might be infected tonsils. Several blood transfusions were given to bring his condition up to that of a better operative risk, a very difficult procedure because of the cardiac dilatation.

An ileostomy was done by Dr. Claude Hunt at the Research Hospital and through the permanent artificial anus thus created most of the feces has since been evacuated. Daily injections of 2 per cent mercurochrome solution were introduced into the artificial opening and allowed to pass through the colon, voided by the rectum. Tonsillectomy was also done. Six months later the boy's hemoglobin was 90 per cent and his weight greatly increased, the appetite good, the contractures of the knees disappeared. He was able to walk without any disturbance, but because of his heart lesion no violent exercise was taken. Apparently he is practically well of the dysentery.

**Treatment.**—Removal of infectious foci in the tonsils, teeth or sinuses should be done, although the benefit therefrom may be only temporary. It is possible, however, that the original lesions may be caused by some such focus and in the future it will be my practice to *remove foci of infection in the very beginning of the disease.*

The difficulty of getting the child to eat adds to the problems of treatment. There should be no starvation period in the handling of these cases. An easily digested food which appeals to the liking of the child is to be given in as large an amount as he will take. The food should contain

fresh fruit juices. One child was fed all the ripe banana he would take. It was well borne and helped to increase the body weight. Banana is so high in food value that it offers a great nutritive addition.

Because of the constant hemorrhage and loss of fluids by the bowel this disease calls for repeated blood transfusions. In addition hypodermic injections of physiological salt solution as much as a pint every other day will be of great assistance in supplying fluid lost by the bowel and in overcoming the insufficient intake by the mouth.

The secondary anemia should be treated and its progress arrested by the daily subcutaneous injection of soluble citrate of iron one or two grains. The broth from iron-containing vegetables is of value.

Many kinds of oral medication attempting to affect the diarrhea and intestinal hemorrhages were used in the three cases which were under my care. Remedies which are ordinarily used are without avail, such as bismuth, charcoal, tannic acid, and emetin. Paregoric and other opiates may give some relief from pain and excessive peristalsis, and should be administered where suffering is extreme. There is no benefit from anti-dysenteric serum intravenously or intramuscularly. The rarity of the disease in children gives too little opportunity for investigation of any one remedy which might prove of value. Two drugs by the mouth seem to offer some hope, in the experience of several observers during the last few years. These are large doses of tincture of iodine, as suggested by Logan, and kaolin also in adequate amounts. One of my cases was given the iodine preparation in syrup over a period of several months. Helmholtz recommends the use of kaolin in 15- to 30-grain doses for short, repeated periods.

An autogenous vaccine has been tried in several cases, with marked benefit in some instances.

Early surgical treatment offers the best outlook for cure. Operative interference is too long delayed, and usually not thought of in these cases until a protracted course and repeated examination reveal the fact that the disease is not acute dysentery, amebic diarrhea or typhoid fever.

The principle involved in operation is to give rest to the diseased portion of the intestine by interrupting its function. Therefore an intestinal fistula is made in the abdominal wall at a two-stage operation. The appendix, ileum, cecum or first part of the colon may be a portion of the intestine utilized in the operation. The Brown operation utilizes the ileum with both ends of the loop available for thorough irrigation. The proximal end of the loop voids the feces and little reaches the distal portion and colon. The inflammation and ulceration of the colon disappears in favorable cases. Most of the bowel contents pass from this artificial opening. Apparently peristalsis and tenesmus in the colon are greatly reduced by this method. A movement from the rectum occurs infrequently so that the amount of blood lost becomes at once markedly reduced. Pain is also much diminished. Nutrition improves and with it the appetite.

As soon as the diagnosis is made, irrigation of the large intestine should be begun. This can only be successfully accomplished through an artificial anus resulting from an appendicostomy or cecostomy. When the disease lasts two or three months without any remission and with progressive anemia and emaciation, the case should at once receive surgical care.

*Summary of Treatment.*—The handling of chronic ulcerative colitis is best accomplished by the following routine:

1. Removal of diseased tonsils, carious or infected teeth, drainage of infected sinuses
2. The oral administration of tincture of iodine in large doses, alternating with large doses of kaolin
3. Transfusion of blood from healthy suitable donor
4. Injection with autogenous vaccine
5. Plenty of digestible food
6. Operation by producing an artificial opening into the ileum for drainage of feces and irrigation of the remainder of the large intestine

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## CHAPTER XVII

### CHRONIC INTESTINAL INDIGESTION AND CELIAC DISEASE

#### CHRONIC INTESTINAL INDIGESTION

Many children from two to five years of age and occasionally somewhat older have a tendency to frequent digestive upsets. Such cases are often regarded as due to chronic intestinal indigestion. There are present, however, in this condition no known lesions of the intestines other than dilatation and a reduction in the muscular tone. This undertonicity is not confined to the small intestines where digestion chiefly takes place, but the large bowel and the stomach share in the chronic dilatation, and the abdominal walls also are flabby. As a result the motor function of the gastro-intestinal tract, as regards peristalsis and drainage, is found definitely impaired.

So-called chronic intestinal indigestion is therefore a functional disturbance and not a primary disease of digestion. There are many causes for this digestive insufficiency. Some children seem to be predisposed by frailty or a "delicate constitution" which has existed since birth. Digestive disturbances are common in the neuropathic child. Hereditary syphilis and cardiac disease are the underlying factors in other instances. I have not found that feeding disturbances during the first year leave any trail of digestive difficulties in later life, unless the child stays markedly under-nourished and continues to be improperly fed after the age of infancy.

All children with persistent digestive disturbance should have a complete physical examination. The diet which the child is accustomed to take should be ascertained. It is apt to be incomplete and to consist almost entirely of starchy foods, sweets and milk. In some cases fats are not tolerated. However, there will be found other factors which are just as important, and many of these are outside of the intestinal tract. Frequent or chronic infections, during the time that they are present, interfere with the digestion and utilization of food. The most common illustration is that of the child who has repeated "colds" from autumn until summer. Frequent attacks of influenza, one contagious disease such as measles may quickly follow upon another such as whooping-cough, and there may be left complications of chronic otitis media, latent mastoiditis, diseased tonsils, sinusitis, cervical or bronchial adenitis, or bronchial fibrosis. These diseases may not be apparent, but the appetite and the stools remain abnormal, due to moderate fever and toxemia. It is not infrequent for pyelitis to run an extended course, the infection undiscovered, and result in chronic intestinal dis-

turbance. That such infectious states are the cause of functional indigestion is proven by the promptness with which intestinal symptoms disappear and health is regained when once toxemia is eradicated.

Occasionally the child is known to have an idiosyncrasy for certain foods. The mother's statement regarding such a peculiarity should receive due consideration, and offending articles of diet excluded. However, it is not at all rare to find that the child's dietary has been immoderately reduced until there is scarcely anything left for the child to eat, because of the supposed general digestive intolerance. This is of course a mistaken notion, as there is never such a child who cannot digest most of the simple well-prepared foods.

The symptoms will depend upon the underlying cause. The only ones which are dependent upon the functional disturbance in the bowel are impairment of appetite and the abnormality of the stools. Constipation is the rule, but there may be periods of diarrhea which seem to be easily provoked by foods which are in excess of the digestive ability, but which in normal children cause no difficulty. Occasionally there is the passage of abnormal amounts of mucus, but this is, in my experience, rare, and is often due to other causes such as intestinal parasites, or a mucous colitis in a highly neuropathic individual. The general symptoms which one finds are those of any nutritional or chronic infectious state. Poor appetite, underweight, flabby muscles, intestinal dilatation are nearly always present, and if the condition is a severe and long one the child may be retarded in growth. Children with chronic dyspeptic disturbances show the same tendency as adults to a persistently peevish disposition, requiring tact and firmness in their handling. These children do not do well in poor environment, *i.e.*, neuropathic, irritable parents who either produce a marked nervous reaction in the child, or who have no control or discipline in the management, especially as to the feeding. It is not the symptomatology of the gastro-intestinal tract which is so important as those symptoms which disclose the actual cause of the disturbed function. These should be determined and properly treated. When there is intolerance for carbohydrates and fats, and the stools are fatty, the case is more serious and belongs to the type next considered; namely, celiac disease.

**Treatment.**—The best results will be obtained by treatment along broad lines.

Of first importance is the removal of gross causes. From what has been said the necessity for eradication of infectious foci is evident. Safeguarding the child from epidemic disease is important but is difficult. Infections of the teeth, the sinuses, the ears, the tonsils, the glands and the thorax should receive attention.

The hygienic care of the child will give beneficial results. During the outdoor life of the summer these children have little difficulty in staying well, and this indicates the value of fresh air, sunshine and exercise through-

out the year. Attention to the regular emptying of the bowels is always to be maintained. It is better not to give laxatives, but to depend upon correction of habit, diet and then if necessary the use of a suppository. Such children do better if they have at least twelve hours of rest and sleep and also a midday nap.

The dietary régime consists of three or four simple meals daily. The variety should be sufficient for the needs of the growing child, but should not be profuse. The quantity can safely be left to the appetite of the child if the foods are simple and not rich. Fats should be carefully regulated, and the amount allowed small until the tolerance is determined. Fried and greasy dishes are never well borne. The same caution should be used in the feeding of starches and sugars. A well-cooked cereal once daily will be digested. Excess of sugar may cause fermented stools. One pint of skimmed milk, sweet or soured, is enough in the beginning of the treatment, but this amount of whole milk may be given after improvement has begun. Steamed vegetables, cooked fruits, fruit juices, dry bread, lean meats, gelatin and many other unobjectionable articles will make up the total dietary.

Medicinal treatment is of limited value. Strychnin is probably the best, because of its tonic effect upon the appetite and the intestinal motility. There is no advantage in giving cod-liver oil in these cases, and it may even be harmful if there is an intolerance for fats.

For the child who has intestinal dilatation and ptosis, the digestive functions will be improved by the wearing of a corset belt.

Children do not die from so-called chronic intestinal indigestion. In fact, the outlook is good for the transformation of these irritable, under-nourished individuals into happy normal youngsters, provided discipline, proper treatment and carefulness in diet be maintained.

## CELIAC DISEASE

### *(Infantilism with Fatty Stools)*

Celiac disease presents a very definite clinical picture beginning in the last months of the first year or during the second year of life. It may persist for several years. After seeing one case, recognition of others becomes easy. The characteristic symptoms are the persistently enlarged abdomen, the marked disturbance in weight, the retardation of body growth, the daily tendency to numerous bulky, light gray, mushy, rancid and foul stools containing much fat and fatty acids. The disease is markedly different from any other disturbance involving the digestive tract of children.

**Nomenclature and History.**—It is now thirty-seven years since this disease was described by Gee, who gave it the name of celiac disease. This term is not descriptive as the word "celiac" means simply abdominal. In 1903, Cheadle discovered that there is an excess of fat in the stools. Among

the other valuable British contributions have been the numerous observations by R. Miller, John Thomson, Gibbons, and Bramwell. Herter, in America, published a monograph in 1908 upon the subject of infantilism with chronic intestinal infection. His opinions have been widely read and quoted and the disease which he described is identical with celiac disease. Recently Reginald Miller has reviewed the nomenclature and has concluded that no name thus far suggested is as appropriate as celiac disease. In few textbooks thus far has there been any mention of the disease as we now know it. It has been included under the name of chronic intestinal indigestion. There have been numerous synonyms, such as pancreatic insufficiency, pancreatic infantilism, and acholia. Because of the two principal characteristics of the disease, namely, the retardation in body growth, and the wastage of fat in the feces, it would seem that a term which well describes the condition is *infantilism with fatty stools*. The name celiac has recently been widely used and is probably here to stay.

**Etiology.**—The disease is found in children under five years of age as a rule. The history points to its inception after weaning from the breast. Most cases date to the age between nine and eighteen months. By the time the physician sees the child, the disease may have lasted several years.

There is little difference in the sex incidence; possibly more girls than boys are affected. Most writers state that the children of the well-to-do furnish the greater number of cases.

Little is known about the essential cause. In most instances the infant has been normal during the nursing period; therefore it seems improbable that there is congenital origin.

Cheadle thought the disease due to a deficiency of bile entering the intestine. There probably is some reduction in the amount but it is not absent in the stools. The bile salts in the duodenal juice are normal.

No constant change in the pancreas has been found, although numerous writers have thought there might be a deficiency of pancreatic secretion. This is not probable, especially in view of the fact that the disturbance is not one of fat digestion, but of assimilation. In 1902, Bramwell used the term pancreatic infantilism in describing a case which had undigested fat in the stools, a case clearly due to deficiency of pancreatic juice. According to present opinion, no pancreatic dysfunction occurs in celiac disease. The pancreas is usually found normal at autopsy.

The feeding of too large an amount of fat and of improperly cooked food has been thought to have a place in the etiology. It is not probable that this is the case. Some cases seem to begin insidiously while others have had prolonged digestive disturbances. When the disease occurs in children who have always been carefully supervised, it is convenient to presuppose the factor of congenital weakness. There are infants whose digestive tracts are able to handle breast milk, but at weaning time are intolerant of artificial foods, at least in the manner that they are sometimes fed.



Quite a percentage of cases will give a history of summer complaint or dysentery. If these have anything to do with causing and prolonging the disturbance in assimilation of food, they leave no anatomic trace that has been recognizable in the intestinal tract. It is probable that in some instances the so-called preceding attack of diarrhea is really the first symptom of the actual disease.

Herter found in the stools of these children an overgrowth of Gram-positive microorganisms similar to those of early infancy, and he believed that these bacteria with their toxins induce a chronic inflammation of the intestinal wall, which interferes with the absorption of food. It is probable that there is an abundance of fermentative organisms in the intestinal tract, which results in excessive fermentation and intolerance for the carbohydrates of artificial foods. If peristalsis were increased one would expect the fats to be hurried through the intestinal tract without time for digestion. However, the fats are well digested. Chronic pathological changes as described by Herter are not found in the intestinal mucosa. Therefore his theory is not regarded as proven.

**Physiology and Pathology.**—What change there may be in the physiology of the duodenum is not yet determined. In one case recently reported by Davison, the enzyme strength (amylase and trypsin) of the intestinal contents was found to be much lower during the periods when the symptoms are pronounced than when clinical improvement occurs. Cautley found a slight increase in the diastatic activity of the stools and a striking absence of urobilin. Celiac disease is a failure of the child to absorb fat. The fat is digested, but is excreted in abnormal amounts as fatty acids and soap. Intolerance for carbohydrates interferes in some way with the assimilation of fat, so that both these foods are poorly utilized. Sugar will not be found in the urine, although one such case has been reported. Proteins uncombined with other food elements are well tolerated.

The pathological changes that are constant seem to be the effect and not the cause of the disease. The intestine, both small and large, especially the colon, becomes greatly dilated. This is due to excessive fermentation of sugar and the resulting tympanites. The abdominal wall becomes atonic, allowing still further distention of the intestines. As to essential or primary changes connected with the disease there are no constant findings. In one case of the author coming to autopsy, the liver showed fatty degeneration, the intestines a follicular enteritis of mild degree, and the pancreas a fibrous proliferation. These may have been due to toxic or infectious complications and not to the celiac disease itself. However, fatty liver has been reported in some cases, while in others the liver has been found to be normal, in some larger than normal, in others smaller.

**Symptoms.**—The disease usually begins after six months of age, most frequently in the last quarter of the first year or in the first half of the

second year. An attack of diarrhea may be the first characteristic symptom. It is soon noticed that the child does not tolerate the usual artificial food. Cow's milk especially upsets the child and causes stools which are characteristic. They are peculiarly bulky, larger than would be expected from the amount of food taken. This is due to the lack of food utilization. The odor is particularly offensive, and so penetrating that it is noticeable at a considerable distance. The stool is foul, sour, musty and resembles rancid butter or cheese.

The *stools* are usually light gray or nearly white. They may be formed, but at times are diarrheal, when they will contain some undigested material, and much fermentation. There are more stools than normal. The composition is largely fat, as high as 70 to 90 per cent, which is three times that excreted in a healthy child. There is little loss of protein by the stools. It is probable that increased amounts of calcium are lost by the stool. Bile is present, though it does not give evidence of sufficient amount to cause a characteristic color to the feces. The general appearance of the feces during the active period of the disease is that of porridge. At times the stools appear greasy.

As the affection proceeds the *nutrition suffers*. There is much fluctuation in weight with a net result of much loss, often to one-third or one-half of the normal for the age. There is little body fat, the muscles become flabby and atonic. This is especially noticeable in the chest and extremities. The arms and legs are thin and small. This wasted appearance is in marked contrast to the ponderous abdomen.

Nutritional impairment is further shown by the slow body growth. The child becomes stunted so that the height may eventually be as much as 6 to 12 inches below the average. The bones are small and, according to Still, the ossification of the cartilages is delayed. The retardation in growth has given rise to a term that is frequently employed in this disease; namely, infantilism. It resembles in this feature the retardation found in diabetes mellitus. The imperfect metabolism of fats and minerals with their marked wastage in the stools undoubtedly causes the delay in skeletal growth. The longer the disease has lasted the more noticeable is the stunting of the child.

The *abdomen* is markedly enlarged, usually one-third above the average circumference. This is due to the dilatation of the intestines, particularly of the colon, which may be several inches in diameter. Tympanites and flatulence are evident; the abdominal wall is thin and may show venous engorgement. The size of the abdomen varies from day to day and is greater in the daytime than at night. Abdominal discomfort from distention is present at times, but there is no evidence of real pain.

The foregoing symptoms are the characteristics by which the disease is diagnosed. Let us now consider the other manifestations which are common

but do not belong exclusively to this affection. The child is sallow, and shows pallor of the skin and mucous membrane. This is due to a reduction in hemoglobin rather than in the red blood-cells, which are less affected. There is nothing characteristic in the blood-picture except a diminution in the hemoglobin. Fatigue is brought on by even moderate exercise; the child sweats easily. Changes in the disposition are noticeable and management is difficult. The child seldom smiles and cannot be pleased. *Irritability*, whining, crying and other nervous symptoms are present during the active periods of the disease. The child is restless during the day and the sleep is disturbed at night. Convulsions may occur, probably associated with tetany. Irritability is due to ill health and starvation. These children are bright and show no brain impairment.

One of the features which interferes greatly with the handling and cure of the case is the poor appetite. At times the child does not wish any food and it is particularly difficult to get him to eat the kind that must be offered him. As soon as improvement takes place, the appetite is less disturbed and at times may be ravenous.

Fever is usually absent during the disease. A few degrees are often present when the child undergoes diarrheal exacerbations or complications.

The urine is much reduced but varies with the intake of food and the amount of fluid lost by the bowel. The fact that it contains indican is of no diagnostic importance. Albumin may at times be found. Examination of the urine should be made at weekly intervals as a matter of routine.

Occasionally the extremities may be edematous, and peritoneal fluid has been reported in a few instances. There are



FIG. 37.—CELIAC DISEASE

Note the large size of the abdomen, the undernutrition of the child and the unhappy facial expression.

no symptoms referable to the heart, lungs, liver or spleen.

The following case reports are of children, one of whom has recovered and the other has died.

I. J. T., one-and-one-half-year-old boy, was under observation in the Kansas University Hospital for four months. He had been sick previously for three months following an attack of so-called summer diarrhea. The stools had been loose and the body weight steadily decreasing. He entered the hospital with a provisional diagnosis of either Hirschsprung's or tuberculous mesenteric disease.

His weight was 15 pounds (40 per cent below the average for age), his height 32 inches (at this time about normal), but later showing marked retardation, his nutrition and body strength extremely low and apparently past remedying. It was difficult to get him to eat, he vomited occasionally, the abdomen was greatly enlarged, the disposition was peevish and the child slept poorly.



FIG. 38.—GREATLY ENLARGED COLON IN CELIAC DISEASE

During this time the temperature remained below normal, from 96 to 98° F. The appearance, odor, frequency, size and almost pure fatty content of the stools established the diagnosis. The blood showed a secondary anemia, including a reduction of the leukocytes to 5,900. Because of the starvation period which this child had been undergoing, a study of the blood chemistry was made. The blood-sugar storage was only 55 Mg. as compared with 100 Mg. per 100 c.c. of blood in the normal child. His retention of urea nitrogen in the blood was



also low, 4.2 Mg. as compared with the normal average of 12 or more Mg. per 100 c.c. of blood.

Treatment was initiated by the transfusion of 300 c.c. of compatible blood. The diet suitable for celiac disease was well tolerated and during the six months which followed the stools became brown, of better odor and the body weight increased to 24 pounds, a gain during treatment of 9 pounds. One year later no relapse had occurred. The diet was still carefully supervised, the body fat and weight normal, but the child undersized.

2. John B., aged four years, had a two-year history of enlarged abdomen, recurrent diarrhea and poor appetite. The disturbance in his health followed influenza and whooping-cough. He usually had from three to six foul smelling stools a day, with periods of improvement when fed on a suitable diet. He entered the hospital in a poorly nourished state, the extremities emaciated, the skin hanging in folds, his weight 28 pounds (about 7 pounds below the normal average). His height  $38\frac{1}{2}$  inches (about  $1\frac{1}{2}$  inches below normal). The urine was normal and the blood showed a slight reduction in the hemoglobin.

The child remained in the hospital about three months. During this time the diet consisted of sour protein milk, gelatin, cottage cheese and well-ripened banana mash. He was given occasional doses of castor oil and a soda enema daily. In the course of two months a gain of 3 pounds was made, the appetite and general condition of the child apparently progressing favorably, when he developed a respiratory infection. This was for two weeks afebrile and limited to the upper respiratory tract. Pneumonia developed and the child died a week later.



FIG. 39.—CELIAC DISEASE

After six months' treatment, the nutrition in this case of celiac disease is much improved, but the abdomen still too prominent.

At autopsy the stomach was found enormously dilated, as were also the intestines. There was a constriction in the first portion of the duodenum, evidently due to the traction from persistent cystico-duodenal fetal bands. Peyer's patches and the lymphoid follicles in the ileum were somewhat congested, the mesenteric glands were hyperplastic. The liver showed some fatty degeneration. The pancreas and biliary passages were normal. The lungs showed bronchopneumonia of no great extent. Nothing in the autopsy could explain the cause and course of the celiac disease.

**Course.**—With early treatment, proper feeding and no complications, the disease may disappear in the course of six months. The disturbance may have existed for months or

a year before its nature becomes recognized. The course is chronic and characterized by relapses and periods of improvement.

The child shows first an improvement in the appetite and disposition. He loses his peevishness and becomes cheerful. This may require several weeks or months. The next indication of convalescence is found in the

stools which acquire a more normal color, consistency and odor. A reduction in the number of stools is finally accompanied by a gain in weight. Marked fluctuation in gain and loss of weight is characteristic of the disease.

Unfavorable cases fail to show any recuperative tendency. Their nutrition fails completely and death results from starvation, asthenia, or intercurrent disease, particularly pneumonia.

**Complications.**—Exacerbations may occur at various times in the course of the disease, during which the stools are diarrheal, the temperature above normal and the loss of weight marked. They are due to mistakes in the diet as to quality and quantity and sometimes to causes not discoverable. Exacerbations are frequently regarded as complications but they can hardly be said to be such. Infections of the nose, throat, bronchi and lungs are frequent and are due to the lowered resistance. They interfere profoundly with the welfare of the case. Scurvy, purpura, urticaria and edema of nutritional origin are found in some instances, either dependent upon the deficient diet or as a complication of the disease.

**Diagnosis.**—The clinical picture has only recently been separated from the class of chronic intestinal indigestion. It is usual for the greatly enlarged abdomen to attract first attention and there comes into the differential diagnosis the possibilities of *Hirschsprung's disease*, *tuberculous peritonitis*, and the *pot-belly of rickets*.

The history will be of much help in *Hirschsprung's disease* because it exists from birth. Constipation exists for days at a time. There are some points of similarity. The abdomen is larger and the radiogram of the colon shows an even greater circumference than in celiac disease. The child is peevish during periods of toxemia which are accompanied by fever and fecal impaction, while in celiac disease fever occurs only during diarrheal exacerbations or complications. Miller believes that there is definite evidence connecting celiac disease and megacolon.



FIG. 40.—SEVERE CASE OF CELIAC DISEASE SHOWING EXTREME WASTING  
The skin hangs in folds. (Courtesy of Joseph Brennemann.)

In *tuberculous peritonitis* the abdomen is enlarged, not from intestinal dilatation but from the increase in the size of the mesenteric lymph-nodes, from an accumulation of fluid (ascites), or from an inflammatory fibrinous exudate. Masses of glands may be felt through the abdominal wall. There is usually some elevation of temperature. The skin reacts to the tuberculin test. Tuberculosis may be found elsewhere in the body. The abdomen steadily enlarges and shows no remission in size, while in celiac disease there are fluctuations from day to day, and the size is smaller at night than during the daytime. The stools are constipated, except when intestinal ulceration occurs, following which there may be diarrhea with bloody and purulent discharge.

In *rickets* there is enlarged abdomen but not to so great an extent. The active symptoms do not persist after the third year. Constipation ordinarily alternates with diarrhea. Perspiration is excessive but is usually most marked upon the head. The rachitic child is not emaciated. Deformities of the bones are evident. It is possible for the diseases to coexist.

Amebic and bacillary dysentery and chronic ulcerative colitis are characterized by bloody or purulent stools. Ulcers are present in the rectum. The abdomen is not enlarged. In amebic infection the causative parasite, *Entamæba histolytica*, is found in the stools.

A white diarrhea occurs in *sprue*, a disease of the tropics. The stools are much similar to those of celiac disease. There is, however, catarrhal inflammation of the intestines, as well as a catarrhal stomatitis.

**Treatment.**—In the handling of this disease, it should be borne in mind that we are dealing with one of the most chronic affections in childhood and one in which there is no tendency to spontaneous recovery. Two chief problems are present throughout its long course; one is the adaptation of the food to the proper kind and amount, and the other problem is to get the child to take these and leave all other food alone. In addition to the absence of appetite, the feeding difficulty is increased by a dietary to which the child has not been accustomed and most of which happens to be more or less distasteful.

In the beginning it is well to inform the parents of the difficulties in the treatment, the dangers of relapse and the long duration, and that if the dietary rules are broken, disaster will occur. The instructions given to the parents must be rigidly followed by them. This includes the maintenance of discipline for the child. Treatment in the home is attended with difficulties. There are many opportunities for the child to be given what it wishes rather than what it needs. It is usually the case that the proper nurse can get the child to eat better than can any member of the family. Short periods of supervision in the hospital will be advantageous.

In the beginning it is well to clean out the bowels with a dose of castor oil, and this may be repeated at intervals, once or twice weekly. Because of the sour and offensive stools and the tympanites a high enema should



be given daily. For this purpose one level teaspoon of bicarbonate of soda is added to a quart of water.

*High Protein Diet.*—Protein alone is well tolerated. It must furnish the basis for the dietary. Other food elements which are essential for well-being must be started slowly and in small amounts. It is well to separate the feeding of these cases into three periods, as suggested by Howland. First, during the worst stage of the symptoms, a high protein food with little carbohydrate is the sole article of diet. Next, as soon as improvement begins, more protein is added, as is also carbohydrate in the form of banana flour or banana mash, a limited amount of fruit juice and cod-liver oil. The third stage is the period of convalescence during which a strict but somewhat larger variety in diet may be given under careful supervision.

It is not possible for the child to thrive on pure protein alone but proteids as they occur in naturally or artificially soured milk are well tolerated and are sufficient to sustain the child until improvement begins. Fat-free buttermilk, either of the old-fashioned variety or soured with Bulgarian organisms, should be the first food used, beginning with small amounts of 1 pint in twenty-four hours and increased gradually. To this may be added dried casein, from 1 to 3 ounces daily, as suggested by Marriott. There are various preparations obtainable, among them casec, larsan, etc. As an alternative to dried casein, fresh curds may be used. These are obtained from a quart of fat-free buttermilk, precipitated by hot water.

Instead of beginning with a fat-free buttermilk, many physicians use a protein milk powder such as the preparations of Merrill, Mead-Johnson & Co. or Hoos. The amount needed in twenty-four hours is 1 packed level tablespoonful for each pound of the child's weight. If the child weighs 12 pounds, 12 tablespoonfuls are added to a quart of boiled water at a temperature of 100° F. Sauer has suggested a method of feeding protein milk which may be used by way of variety. He makes a thick paste by adding a small amount of water to the milk powder. Instead of plain water, he adds a tablet of Ringer's solution so that the child gets the added salts which he believes help in the better absorption of the food. This paste is flavored with vanilla and sweetened with saccharin and one or two feedings are used daily.

The child should be fed only three meals a day and the quantity of the food should be commensurate with the ability to handle it. It is better to begin with half of the amount which is theoretically indicated and increase gradually. Overfeeding is responsible for some of the upsets which are so liable to happen. With the subsidence of diarrhea, the diet should be increased by the addition of accessory food substances containing vitamin C. Orange juice is the safest and most advisable, as it usually has no laxative effect and is a diuretic. Later vegetable juices may be tried in small amounts, so as to supply the mineral salts. Some difference of opinion exists as to the tolerance of cod-liver oil. Vitamin A is needed



in the relatively poor diet which these children receive. Cod-liver oil, pure or as a powdered tablet of the extract may be begun once daily, and increased slowly.

In the second period of the dietary treatment, it is necessary to furnish a larger variety as the child gets tired of the monotonous diet. Cottage cheese made from skim milk is well borne. Gelatin, flavored with fruit juice or meat extract, is well liked.

In health the body tolerates large amounts of carbohydrates, fully half of the total calories being thus supplied. In celiac disease the tolerance for sugars and starches is low, and only one-fifth of the total food can be fed as carbohydrate. From the experience of Haas and from a limited number of personal cases the author believes that the feeding of well-ripened banana may be begun early in this disease. This food makes it possible to use an adequate amount of carbohydrate. Banana owes its food value chiefly to the amount of sugar which it contains. In 1 ounce (30 grams) of banana there are 15 grams of sucrose (finally changed to invert sugar), 1 gram of protein, and  $\frac{1}{2}$  gram of fat. The caloric value is three times that of milk. The banana should be thoroughly ripened as is found when the yellow skin begins to change to brown or black. The prejudice against banana feeding in childhood has arisen from the undigestibility of the unripened fruit, due to its unchanged starch. Finkelstein and others of the German school have for many years used banana in the dietary of children. It is well handled not only in normal children, but in nutritional disturbances and in celiac disease. Haas believes that it may be fed in large amounts at the very beginning of treatment. It is well, however, to use it with caution at first until its harmlessness in an individual case is proven. The technic of preparation is to put the well-ripened banana through a colander and the child is allowed to eat with a spoon. It is always well liked and the child will take almost unbelievable amounts, sometimes from six to twelve bananas daily. The following table will give an idea of the calories received by a young child that made a complete recovery, and this child did well on the high caloric feeding:

DIETARY IN CELIAC DISEASE IN THE CASE OF C. B.

Week	Average Daily Calories	Protein Milk, Calories	Bananas, Calories	Body Weight
1st week .....	1,089	...	298	15 lbs.
2nd week .....	841	439	402	16 lbs.
3rd week .....	1,083	475	608	16-6 oz.
4th week .....	1,260	570	690	16-14 oz.
5th week .....	1,290	600	690	16-14 oz.
6th week .....	1,290	600	690	17 lbs.
10th week .....	1,520	600	920	18-14 oz.

Although these cases require from two to three times the usual number of calories per pound in order to gain in weight, the tolerance and well-being

of the individual are of first consideration. The physician should be satisfied with general improvement and slow gain in weight, rather than incur the risk of diarrhea from overfeeding.

The foregoing strict diet should be used for several months. The third period of feeding is begun as soon as convalescence seems established. In addition to the previous foods, the child may be given vegetable soup, stewed fruit without sugar, scraped lean beef, lamb or tongue, white of egg, well-cooked gruel, toast and arrowroot crackers. The diet must be restricted for several years.

*General Treatment.*—Fresh air and sunshine are as important in this disease as in all others which incapacitate the child for long periods. Baths, massage, and general hygienic measures are of value in adding to the comfort and welfare of the individual.

*Medicinal Treatment.*—Iron citrate or lactate,  $\frac{1}{2}$  to 1 grain three times daily, is well tolerated and is needed for the anemia. Dilute hydrochloric acid has been tried, but the administration of acid milk is more serviceable.

*Prognosis.*—Early recognition of the disease, appropriate diet, and supervision of the child for many years offer the best chances for recovery. The child will furthermore be spared the nutritional losses and the reduction in resistance to intercurrent infections so commonly seen in unsuccessfully treated cases. One of the most important factors in early cure is the prevention of stunting. Little is known as to whether these individuals attain a normal height in later life.

Holt and Howland state that those cases which have been initiated by faulty diet and unhygienic surroundings are more apt to recover than are the children who have had better conditions of living. This means that the latter class may have some underlying constitutional weakness.

For a short time at least the child should be hospitalized, so that discipline, regular habits of eating, and a regulation of the diet may be established. During a period of close observation, the physical condition of the child, and the tolerance for food may be ascertained. This preliminary stay in the hospital favorably affects the prognosis.

Prolonged hospitalization has the disadvantage of keeping the child too closely housed. Anemia which is already present is increased by lack of sunshine. It is difficult to keep the hospital child away from contact with infections. Both the family and the child are better satisfied in the home. Results there will depend upon intelligent and faithful coöperation, whereby the child can get better individual care. At intervals the child should be returned for observation to the private room at the hospital under the care of a special nurse.

Until recently the disease has been generally regarded as offering a poor outlook for cure. The advances in the knowledge of the highly specialized diet have made the prognosis more favorable. There is much to discourage the physician and the parents in the long and uncertain course. The mor-

## 218 CHRONIC INTESTINAL INDIGESTION: CELIAC DISEASE

tality rate is high in the untreated cases. As yet there are no comprehensive figures. Of forty-one cases followed by Still, in England, he found that six had died. This is a lower death-rate than has thus far been experienced in this country. In six cases treated by Pipping, three died, and only one reached adult life in good health and development. The good results reported by Haas are encouraging but these cases will have to be followed for a much longer time before final conclusions can be drawn.

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## CHAPTER XVIII

### DEFICIENT APPETITE AND CONSTIPATION

Habitual lack of appetite is one of the most common complaints in the modern child. The parents come to the physician with the familiar story that they are unable to get their children to eat well at the table. When after a physical examination it is found that no diseased condition exists the explanation for the so-called anorexia can be obtained when the habits of the child become known. One class is found to be well nourished and the amount of food consumed adequate. The child drinks a quart or more of milk daily either in the home or with that furnished at school. In addition to this, the appetite is satisfied by "piecing" between meals. The amount eaten in this way is not appreciated by the parents. The other class of children is not so well nourished, and is found to be finicky in matters of diet. Many factors enter into the reason for such abnormality. Deficient sleep and rest with resulting irritability occurs commonly. Excessive activity and excitement often make the child unwilling to give the time necessary for eating. Children easily acquire likes and dislikes for certain foods and these may persist into later life. While it is notable that when left to its own initiative the child will eat all sorts of articles, good and bad, one observes that there is a marked preference at meals for a very limited variety. Any one familiar with young life knows that the child selects from his plate only those things which he likes, and he will usually eat generously of these. The child becomes finicky in the household that has too much to eat and too many delicacies. One still sees in the poor or modest home the old-fashioned boy or girl who sits at the table enjoying a frugal meal of bread, soup, meat and milk.

The remedy lies in preventing all eating between meals, so that the stomach becomes empty and hunger has time to develop. A glass of milk, an ice-cream soda, an apple, sweetmeats, and all other edibles so commonly accessible and indulged in will destroy the appetite for the following meal. Simplicity in the matter of a few wholesome dishes at the table will appeal to the child more than a bewildering variety, and he may usually be allowed to follow his own inclinations. The desirable variety should not be attempted at one time, but from day to day.

The influence of the physician is undoubted when he gets the child's confidence. It is well to find out from the child what foods he likes, and then emphasize the ones which are advisable. Suggestion is of great value, and the coöperation of the mother and child will get results.



## PERSISTENT ANOREXIA DUE TO ASTHENIA

Functional and anatomical weakness is sometimes responsible for continued poor eating. The asthenic habitus is accompanied by a flabbiness of the musculature which affects the gastric function. The stomach is ptotic and its emptying time increased, so that hunger is delayed. The amount of total hydrochloric acid secreted is much less than in normal children. The administration of strychnin, dilute hydrochloric acid and essence of pepsin will be of help in such cases especially during hot weather when the gastric secretions are lessened. An abdominal belt will help these cases. The gastric tone is improved as the general nutrition increases.

**Effect of Disease.**—Anorexia is often the result of obvious diseased conditions in the child. A thorough physical examination is a necessity. Infection is the most common cause. The routine inspection of the ear, nose, tonsils, glands and teeth is a fruitful source of information. Chronic "colds," sinusitis, and chronic bronchitis may greatly interfere with the appetite. The presence of a temperature above the normal is nearly always accompanied by a lessened desire for food. Diseases of the regions above mentioned usually have some elevation of temperature.

Of most concern to the physician is the possibility of tuberculous infection. Anorexia is often the early symptom which calls attention to ill health from such a cause. It calls for a prompt examination of the gastrointestinal tract as well as the chest and entire body.

Constant or recurrent fever from infection of the urinary tract is one of the most common causes of impaired appetite. It illustrates the necessity of recognizing anorexia as purely a symptom which may owe its source to an infection outside of the digestive system.

## PERVERTED APPETITE

(*Pica*)

Children are not infrequently found, especially among those of the runabout age, who acquire the habit of eating unsuitable and obnoxious substances. There is no limit to the list which may be taken. Such a habit is a persistence of the infantile tendency to place all accessible articles in the mouth, while in early childhood it is a manifestation of curiosity. It is paradoxical that a child who objects to certain wholesome foods and who can at will react by vomiting them may voluntarily eat and apparently relish tasteless, dirty and abhorrent substances.

This perverted appetite, known as pica, is a cause of much concern to the parents. While children of lowered intelligence and markedly incorrigible show the worst tendency to this habit, it is seen sometimes in bright youngsters whose mothers have been energetic in proper training. In

mentally normal children, the practice is self-limited, never persisting into the school age. Children with pellagra often have this habit.

The most common substances put in the mouth are those found while playing. These are dirt, sand, gravel, sticks, worms, and sometimes excrement. Not an uncommon habit is that of gnawing the paint from toys and furniture. Attention has been directed by Ruddock and others to the danger of developing lead poisoning in this way. Cases have been reported with typical blue hemorrhagic gums, colic, neuritis, convulsions, foot drop and other toxic symptoms.

The correction of the habit is most important from the standpoint of preventing ill health, malnutrition, infections and poisoning. The ova of intestinal parasites may be ingested, and pathogenic microorganisms undoubtedly reach the intestines in such a way. Restraint is necessary as a prophylactic and as with other bad practices the substitution of a good habit that will occupy the interest of the child. In poisoning from paint, the medical treatment for elimination of lead should be promptly used.

## CONSTIPATION

**In the Newly Born.**—In the absence of obstruction, the well-being of the infant is not affected by the failure of an evacuation on the first day and this need therefore cause no alarm. Meconium is usually passed soon after birth, but if there is none by the second day, an enema should be given. In non-obstructive cases the stools will be established as soon as food has been begun. Inanition in the young infant because of insufficient food is a cause of constipation. In the absence of fever, it is unnecessary to administer any laxative by the mouth.

When there is obstruction of the esophagus or duodenum, vomiting will be associated with the constipation. Congenital hypertrophy of the colon as well as microcolon causes constipation from the beginning of infancy, but is a rare manifestation. Imperforate anus is the most common cause for absence of stool in the newly born. Atresia of the rectum, colon or small intestine is found but rarely.

**In the Breast-fed Infant.**—The infant receiving plenty of breast milk is seldom constipated, nor is the stool hard or markedly formed in the nursling. Failure to have one or more daily stools is due usually to the lack of sufficient bulk for the stimulation of peristalsis. The fat, sugar and protein of breast food are so well absorbed that comparatively little is wasted in the stool. In infants receiving insufficient breast milk the stools are infrequent and small. It is the lack of a movement and not a hard consistency which causes the mother much concern and which brings her to seek advice for constipation. There is, therefore, no mechanical difficulty inherent in the size of the fecal mass which interferes with the passage, as is the case in artificially fed infants having large, formed, dry, milk stools.

Alternating constipation and diarrhea are not rare in the *neuropathic infant*. The lack of stools is due to intestinal spasm which is functional. Such infants vomit considerably and this diminishes the fecal output. Diarrhea is due to hyperperistalsis.

Congenital weakness and intestinal atony reduce peristalsis in many cases. Organic lesions exist in some infants, the most common of which is redundancy in the sigmoid whereby kinking, sacculation and ptosis interfere with emptying of the bowel contents. Dilatation of the colon with atony or with hypertrophy of the intestinal wall is a definite cause, though not common. Spasm or congenital stenosis of the sigmoid region is probably more common than suspected.

The cause of constipation should be sought, and every case, no matter how trivial, should receive the attention of the physician. When a mother brings her child for the first time with the history of obstinate constipation, she should be assured that every effort will be made to find and relieve the condition. When an infant has been closely observed from birth a serious organic basis for the obstruction will have been suspected. The examination should include the inspection and palpation of the abdomen for evidence of intestinal *distention* and dilatation, or for the presence of nodules or growths. An inspection of the anus and a digital examination of the rectum should be made. In cases that are clearly obstructive from early infancy, a barium injection and if necessary an opaque meal, are warranted.

In most cases, however, the disturbance is simple. Mothers have a tendency to exaggerate the seriousness of constipation, and often have a preconceived opinion that infants are abnormal if there are not several stools daily.

**Treatment.**—Regular hours for nursing should be established. The amount of food taken in the twenty-four hours may be ascertained by weighing upon accurate scales. It is important to know whether the child is gaining normally. If the underweight child cannot be made to gain on the breast alone, one or more nursings should be followed by a few teaspoonfuls or more of soured milk to which well-cooked cereal has been added. In some cases it is well to include a laxative malt sugar. There is no better remedy for the constipated breast infant than additional food, whether human or artificial. If the foregoing food does not produce a stool, it may be well, in infants beyond four months of age, to give a few teaspoonfuls of fruit or vegetable juice. It is desirable to produce one reasonably soft—not a watery—stool daily.

Much can be accomplished by regular attempts at movements. At a definite hour twice daily the abdomen should be massaged by the mother or nurse, and the infant then held upright upon the vessel for a few minutes while awaiting the stool. There is no objection, while regular habits are being formed, to the use of the soap stick or suppository, if these are necessary.



If, after several weeks' trial of the foregoing methods, the results are not satisfactory, agar with or without mineral oil is of value, given in teaspoonful amounts, once or more daily. These act mechanically and without irritation or pernicious habit formation.

Infants in whom constipation is due to anatomical abnormalities should have in addition to the foregoing a daily high injection of oily and soapy water. Acute obstipation from inflammatory lesions, kinking, invagination, appendicitis or complete obstruction demand prompt surgical relief.

**In the Artificially Fed Infant.**—Constipation in the bottle baby is usually functional, and in the beginning dependent upon the nature of the food. Certain of the prepared foods, notably condensed milk, are so readily absorbed that little remains to form a stool. The stool is usually small, sour and is in some cases dry and crumbling. Such food is low in protein and calcium and produces body fat at the expense of muscle. As a result the nutrition of the intestinal and abdominal musculature suffers, whereby the motor functions connected with defecation become decreased. Pot-belly and dilatation of the intestine result from the gaseous distention. Peristalsis is ineffectual because of the intestinal atony. Constipation is a forerunner and an accompaniment of nutritional disturbances.

Constipation due to cow's milk feeding is very common. It is often incorrectly ascribed to the boiling of milk, but milk which has been gently boiled for five minutes is no more constipating than raw milk given in the same amount. Milk which has been boiled down to a much greater concentration is more constipating because of the higher amount of fat which it contains per ounce as compared with uncooked milk. The stools are, however, somewhat different: with boiled milk the movement is smooth and usually well digested; with raw milk large curds are present. Milk constipation is due to the relative overfeeding of fat, by which the infant's tolerance is exceeded. It results from a lack of balance between the protein-fat content and the carbohydrates. Infants fed upon high protein formulæ without fat can also be definitely constipated when the sugar ratio is low.

Fat constipation is synonymous with the "milk injury" of the Germans, with the "disturbance of balance" of Finkelstein, and the weight disturbance described by Grulee.

The feeding history usually shows that the formula has been increased by the addition of whole milk or cream, or for some time the child has been given high amounts of milk containing much total fat. The infant may have gained rapidly for a while, when constipation, crying, restlessness and a stationary weight for several weeks or months cause the infant to be brought for examination. Laxatives cause no marked improvement in the symptoms. There is usually the complaint that the urine is offensive, which on inquiry will be found due to excessive ammonia excretion, accompanied by intertrigo of the skin in the diaper region. Some infants will have redness of the cheeks, and ultimately eczema of the face, scalp or entire body.



When the symptoms have lasted for some time, nutritional changes are evident in the pallor of the skin and flabbiness of the muscles.

Stools are seldom passed without a suppository, injection or laxative. The consistency of the stool varies. Usually it is a large, formed, smooth mass, glistening and greasy, brown or yellowish. More pronounced cases have dry, hard, granular, grayish-white stools, in detached balls or masses resembling dog stools, which do not adhere to the diaper. When cut they show the smooth consistency of hard butter or putty. The exterior is often streaked with blood. The reaction is alkaline. These stools occurring in fat constipation are composed largely of insoluble soaps. When the constipation first appears the stool is smooth except for small, hard, white, sandlike particles throughout it. The moisture in the feces is absorbed by the intestinal wall. The physician should closely inspect the constipated stool by cutting it with a knife or spatula. The presence of the small grainy substances should suggest at once the necessity for reducing the amount of cow's milk in the formula.

The urine contains a large amount of ammonia, which often proves irritating to the skin, causing an intertrigo in the inguinal folds and over the buttocks. The ammoniacal smell of the wet diaper is one of the first evidences of fat constipation. There soon results a papulovesicular dermatitis of buttocks, scrotum, and neighboring skin, an ulcer of the meatus when the wet diaper touches the skin frequently or for long intervals. When the quantity of milk in the diet is reduced the disturbance is corrected. This does not happen in the breast infant.

The phenomenon of the ammonia-smelling diaper has been ascribed to the influence of unrinsed soap and the use of hard water in the washing. Cooke isolated from the stools of artificially fed infants and from older children who are bedwetters a Gram-positive bacillus which ferments the urine in the soaked diaper and liberates ammonia. He recommends the rinsing of the boiled diaper in a solution of mercury bichlorid. This solution is made by adding a  $7\frac{1}{2}$  grain tablet to each 2 quarts of water in a porcelain bowl, and the diapers after washing are rinsed in the antiseptic solution, wrung out and hung up to dry.

**Treatment.**—In the early stage a reduction in the amount of fat in the food is often sufficient to cause a disappearance of the constipation. Half of the cream is to be removed from the milk used in the formula. It is advisable to sour the milk with lactic acid organisms or with U. S. P. lactic acid, 30 drops in each 16 ounces of cool boiled milk. It is better to begin with 1 ounce of milk per pound of body weight and increase slowly each day as the tolerance permits.

In cases that have been affected for several weeks or longer and in whom the fat injury is severe, it is well to use milk from which all of the cream has been skimmed. Fat-free buttermilk or skim milk is indicated, diluted with equal parts of well-cooked barley or farina gruel. From  $1\frac{1}{2}$  to 2

ounces of such a mixture per pound of weight will be necessary while the fat is being excluded.

Additional sugar is necessary to balance the formula, supply the calories, and to make the stools soft, but not loose. Malt sugar or corn syrup beginning with 1 tablespoonful in the formula and increasing to 4 or 5 daily is well tolerated and produces the best effect upon the stool, which will become brown, salve-like and entirely smooth.

Whole milk may now be gradually substituted for a portion of the skim milk. The increase should be made slowly, giving only one ounce more a day, if the infant remains comfortable, the stools moist and the weight increasing. Infants will gain on low fat if the other elements in the food are sufficiently high. It is desirable to keep the fat out of the food for awhile after the intestinal disturbance has disappeared. In the soured foods such as buttermilk and protein milk fat may be introduced more rapidly than in sweet milk.

The duration of the disturbance and the nutritional state of the infant have much to do with the rapidity with which the food may be increased. Milk injury and intestinal atony have resulted from long-continued constipation of this type, and slow progress is made both in the increased fat tolerance and in the weight. The comfort of the infant and the development of normal stools are of first importance and the weight can then be expected to increase, even on slight increase in the food. Too much emphasis cannot be placed upon the necessity for securing smooth moist stools.

Fruit juices are indicated after the age of three months. It is well not to begin accessory foods during the period that the child is uncomfortable, and the milk formula is being adapted to the individual. These may add to the digestive disturbance and make the formula feeding more difficult. It is advisable as soon as the stools are normal to begin these additional foods. They should consist of one or more fruit or vegetable juices. Orange juice is usually well tolerated, but is not ordinarily laxative, even in one-ounce quantity. I have found that it is advisable to try various ones until the most suitable is found. This may be the juice from cooked prunes, canned tomato, pineapple or canned fruit, beginning with a teaspoonful just before each day feeding, and increasing to a tablespoonful.

When the constipated stools disappear, cod-liver oil combined with malt extract, teaspoonful doses, three times daily, will be well borne. Cod-liver oil is necessary for the utilization of calcium and the prevention or cure of rickets to which in the winter months these infants are peculiarly subject.

Persistent cases develop diarrheal stools while on high milk formulas with large amounts of carbohydrate. Acute colds and other infections are common and may bring on an attack of indigestion or increase the feeding difficulty. In such cases the milk should be withheld for twenty-four hours, and a casein-cereal mixture thereafter used until the digestion is capable of handling small amounts of a low-fat, sour-milk formula.

## CHRONIC CONSTIPATION IN CHILDREN PAST INFANCY

**Definition.**—Constipation is a symptom of disturbed function of the digestive tract, which may be due to causes either within or outside of the intestine. The term means strictly the condensation of the feces to a dryer, firmer consistence, packing the portion of the intestine in which it is contained. The word is also used to indicate a reduction below the normal frequency and quantity of the evacuations. Obstipation implies an extreme degree of constipation.

**Occurrence.**—The condition is a common one in childhood, found at all ages, but not so frequent in older children who can be taught to assist in the efforts to keep the bowels regular, and whose diet is much more varied. While many individuals go through life quite tolerant of constipation, the condition in childhood is more apt to become worse, to cause dilatation of the intestine and prominence of the abdomen, and in long-continued cases, to assist in the disturbance of nutrition.

**Mechanism.**—Intestinal stasis is present in all cases as the mechanical factor. It is caused by a weakness or lack of the normal contractions of the intestinal musculature, due to the influence of habit, insufficient training, or as a result of atony. In other cases there is definite spasm of the sigmoid or anus. It is made worse by the fact that the child fails to respond to normal stimuli, as a result of which peristalsis becomes feeble. Lack of sufficient fecal material reduces the irritation necessary to cause these stimuli. Constipation grows more obstinate with the duration.

Hardness of the stool is due to desiccation by the absorption of water during the stasis, or to insufficient intestinal or hepatic secretion. It may follow the water exhaustion produced by an attack of diarrhea.

**Etiology.**—The causes of chronic constipation are many, the underlying ones being the physical inheritance and the earlier health of the child. In the discussion which follows, the causes will be found to point the way towards prevention and cure.

The most common factors in the production of chronic constipation are: lack of training in infancy, muscular atony, abuse of laxatives, persistent inattention to the bowels, unbalanced diet, undernutrition, postural deformities and visceral ptosis, malformations of the intestinal tract, spastic diplegia.

*Lack of Training in Infancy.*—The causes for constipation should be sought in the early history of the infant. Whether or not the child was normal at the beginning of life, or was always constipated, the lack of proper and regular attention to the excretory function has much to do with the condition in later life. Little effort is made to establish a definite period for evacuation in the nursling. Some, at any hour day or night, pass a stool, while other infants have a somewhat regular hour. It is fairly easy to regulate the time, especially in the bottle baby whose food tends to produce constipation and only one or two actions daily. The mother usually succeeds in



establishing a normal habit when she begins in the early weeks or months. This is accomplished by finding the time which is the usual or most favorable and seeing that the bowel is moved each day at the same hour. This generally happens soon after a feeding. The child is held upon a commode, the abdomen massaged, and until habits are established an oiled glass rod, soap stick or suppository inserted for a few minutes if necessary. It is surprising how soon the effect of training and habit will be shown in the young infant, for by six months of age the child placed upon a chamber may know what is expected of him.

Prolonged diaper usage is a lazy and detrimental habit which encourages constipation by allowing the child to be irregular. The runabout child clad in a diaper is an anachronism. He is unattractive, usually soiled, and is evidence of no attempt at training or discipline.

*Muscular Atony.*—Either from congenital weakness, early constipation or as a sequel of nutritional and diarrheal diseases, the child may emerge from infancy with anemia, flabby muscles, dilated intestines, and a retardation in activity of the digestive tract as well as of the entire body. Rickets is still active during the second year, and its effects may persist throughout childhood. The restoration of the normal body tone is essential to the cure of constipation.

*Abuse of Laxatives.*—It is more common and much easier for the attendants to give laxatives than to spend the time necessary for training the child. Such medicines are well known and are kept in nearly all homes. The mother cannot escape the information in the newspapers and billboards which contain the advertisements of the "best sellers." Laxatives are given for such trivial reasons as teething, irritable disposition, or, because the child is allowed to eat anything he wishes, he is thought to need a "cleaning out." Some mothers give purgatives occasionally as punishment, or once a week on general principles. The effectiveness of the medicine is often estimated by the severity of its action in producing numerous watery stools.

Not only are the parents, the drug stores and the newspapers responsible for this abuse, but we physicians are not free from the tendency to recommend and prescribe laxatives with little real indication. The difference is that castor oil and calomel frequently take the place of the kindlier preparations. The writer is sometimes asked as to what he regards the best routine laxative for children, to which the reply is made that there should be no routine laxative nor an indication for one.

The result of continued purgative administration is that no normal regularity can be established. Instead of a physiological process, the unnatural stimulation by medicine becomes the habit. This eventually produces a lack of response to normal stimuli, and in constipated individuals the tendency is made worse by loss of the normal tone of the intestinal muscles.

*Persistent Inattention to the Bowels.*—Only in recent years has the diet of children been given any considerable attention, and comparatively few



are trained in habits of bowel regularity. The complex program which the modern school boy or girl follows makes it convenient to put off every duty that is not urgent. Late hours for retiring, whether due to the moving-picture show or to the radio, or whatever the diversion, provide too little sleep, and the child gets up so late that there is insufficient time for the hygiene of the body.

The most common factor in neglect is the failure to observe a regular hour, whether the inclination for a stool is present or not. The child should go daily at the usual time. It is well to begin in early life to place the child upon the toilet just after breakfast and again after the evening meal. The young child should use a commode or a seat with provision for resting the feet upon some form of support, so that the legs may aid in exerting abdominal compression. Plenty of time should be allowed, and the child left alone without any distraction. Failure to obtain a stool does not call for laxatives, but for persistence in natural attempts.

*Unbalanced Diet.*—Few families in urban communities live upon the simple coarse diets which were common a few generations ago. The accessibility of sweetmeats and ice-cream preparations, the presence of delicacies, the use of rich and highly seasoned foods, have destroyed much of the desire for plain and wholesome diet. The most common complaint of the parents bringing the child to the physician is that of poor appetite. These children are allowed to eat at any time because so little is consumed at meals. On the other hand, the child may have too obvious supervision at the table. An illustration of this tendency, especially common in the one-child family, is that told me by a woman who said that her twelve-year-old daughter would not eat well. This mother could not understand the apparent lack of appetite, for she said she had always prepared especially every mouthful of food which the girl was offered. The suggestion that the child be permitted to eat the regular table food was acted upon, and resulted in a normal appetite.

Children eat chiefly carbohydrates, such as potatoes, white bread and desserts, and satisfy their further hunger with milk, both at home and at school. Few like the anticonstipating foods, such as bran-containing cereals and bread, and especially fibrous green vegetables. Such "roughage" is necessary to provide the irritation and bulk for stimulation of peristalsis. One reason that so many children do not like green vegetables is often due to the lack of flavor and tastiness. Spinach, canned string beans, and cabbage are not attractive when served water-soaked and poorly seasoned. Spinach may be steamed, seasoned with meat, or mixed with potato, bread crumbs, or meat and baked in croquettes which will be enjoyed by the child.

Orange juice has little if any laxative effect. In children past one year of age the orange may be sliced, and thus the fiber is utilized in the feeding, with more tendency to stimulate bowel movements. Bulky stewed fruits are of value.

*Undernutrition.*—As a rule the well-nourished or overweight child has normal bowel movements, due to normal secretions and functions of the intestinal tract, and to the adequate intake of food and water. The thin child is usually constipated because of the absence of these factors. Thin or flabby musculature affects the entire mechanism of defecation.

*Postural Deformities and Visceral Ptosis.*—Closely associated with underweight and undernutrition is fatigue posture, and nearly all these subjects are constipated. The child with poor muscular development easily becomes fatigued on exertion and reacts by slumping in his posture. The shoulders are stooped, the lumbar curve lordotic and the abdomen prominent. There usually results a ptosis of the abdominal organs in which the colon particularly sags and ineffectually empties itself. In some instances there is a familial tendency to the “lanky” habitus and constipation.

*Malformations of the Intestinal Tract.*—Abnormal mobility in the cecum and colon, redundancy and unusual length of the large intestine cause sacculation and kinking which produce intestinal stasis. Fixation and constriction of the intestine by short mesenteries and by peritoneal bands may interfere with peristaltic function.

*Spastic Diplegia.*—The paralysis of Little’s disease is usually accompanied by constipation. Not only is there paresis of the intestinal musculature but the mental deficiency interferes with the coöperation necessary in securing a stool. Many of these children are unable to sit up, or to bring into play the muscles of the abdominal wall.

**Symptoms.**—There may be no general symptoms. Intestinal toxemia from chronic constipation is rare unless there is marked dilatation of the colon. Constipation is the result of causes which may affect the general health. Fever and headache are sometimes present.

Local symptoms are at times present, such as distention of the abdomen, flatulence, colic. Mucus or blood may be produced by the irritation of the large and hard fecal mass. This causes pain during defecation, and a tendency to withhold bowel movements. Fissures and hemorrhoids are not infrequently caused. When the abdominal muscles are thin, or a congenital weakness of the wall is present, straining at stool may result in the production of hernia. The urine is high colored, reduced in amount, and some indican is present.

The bowel function may be sluggish, with only one stool daily, which is apt to be small and hard. It is common to find the first portion hard, to be followed by a soft portion. In chronic constipation there may be exacerbations and intermittent acute attacks of a severe nature.

Neglected cases show little tendency to recovery and they pass into adult life unimproved.

**Diagnosis.**—Chronic constipation of purely functional character must be differentiated from the mechanical types caused by intestinal malforma-

tion, adhesions, tumors, chronic invagination, etc. The previous history of the case, the appearance of the abdomen and the presence of tumor will be of help. Much of our clinical knowledge of these has been obtained by the roentgen-ray examination, of which the enlarged colon and redundancies are the best examples. Rectal injection of an opaque substance will reveal dilatation of the lower bowel and greatly lengthened portions, redundancies and filling defects which may be strictures or exaggerated kinks. Fecaliths and foreign bodies sometimes cause constipation or chronic obstruction.

They may be felt through a relaxed abdominal wall, or suggested by the fluoroscope and radiogram.

**Treatment.**—The summary of the general principles to be followed is as follows:

*Prophylaxis.*—Constipation may be avoided in most children by proper training during infancy, and the strict observance in later life of regular habits of eating; by the use of a well-rounded dietary suited to the age; by abstinence from laxatives; by regularity in attempts at stool, abundance of fresh air, sunshine, exercise, active play, and the avoidance of digestive and nutritional injuries.

*General Handling of the Case—*

*Diet.*—Most children will do better if

the amount of milk and starches be reduced and the calories supplied with other food. So far as the appetite is concerned milk in large amounts is a self-sufficient diet, in which case it is definitely constipating. The child past infancy can thrive on from 8 to 16 ounces daily, if other lime-containing foods are given in sufficient amount. Buttermilk is better than sweet milk, especially when flatulence is present. Potatoes and rich starches should be stopped.

Carbohydrates are best supplied by well-cooked whole wheat, bran, corn-meal, cereals, bread, graham crackers, honey, sorghum molasses, and malt sugar.

Vegetable purees, steamed vegetables, celery and lettuce are to be used daily. Fresh fruit juices, stewed or steamed fruits should be given at each meal.

*Correction of Posture.*—The wearing of an abdominal corset will greatly benefit children who have a large abdomen and chronic constipation.

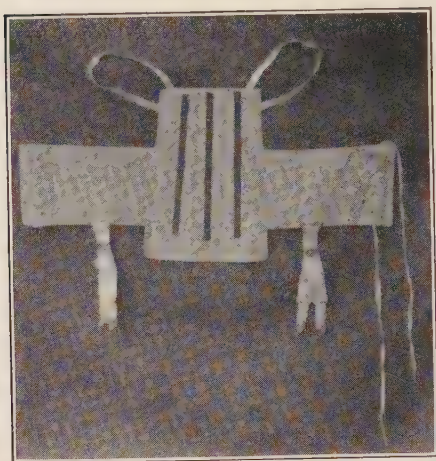


FIG. 41.—HOMEMADE CORSET BELT FOR RELIEF OF CONSTIPATION DUE TO LACK OF ABDOMINAL TONE AND TO VISCERAL PTOSIS



*Massage and Exercise.*—Massage is of value in all cases, especially in the severe ones that have the flabby pendulous abdomen, and no voluntary stool. The mother can be easily taught the technic, if necessary by a few lessons from a masseur. It should be given at the same time each day, preferably at the hour desired for establishing the stool habit. The child lies



FIG. 42.—FRONT AND BACK VIEW OF CORSET BELT IN PLACE

In actual use belt is pulled down farther than is shown in the anterior view and held in place by proper fitting and by attachment of the supporters to the stockings. The posterior portion of the belt should not be long enough to cause discomfort at the upper or lower ends.

on the back with the legs relaxed. The abdominal muscles are grasped and kneaded and by deep pressure the intestine is stimulated, following the course of the colon from right to left. Pressure by numerous mechanical measures has been used, such as by croquet balls and wooden dumbbells. It is unnecessary and unsafe to resort to the "services" of the faddists for massage or passive manipulation.

The simplest active exercise is the outdoor game, in which the child is allowed to play naturally. If crawling or creeping enters into the game the



effect is better. The horizontal bar can be put up in the yard or house and it is popular with all children.

*Hydrotherapy.*—Few children, with the exception of bedwetters, drink much water. There is much benefit from water in constipation, especially if fruit juices are added. Enemas are indicated in cases which are subject to fecal impaction. They are not to be used as a routine.

*Medicinal Treatment.*—Many drugs have been recommended, but few are indicated and these should take a secondary place in the treatment. In the absence of acute toxemia none should be given. The laxative habit is a common one and has no permanent beneficial effect. One of the most striking illustrations of this occurred in the case of a five-year-old girl whose father emphatically asserted that nothing would move the bowels and that he had tried all the available drugs. This child was placed under observation in the hospital, and given a full diet without any laxative agents. Prompt results were obtained, there occurring two normal stools daily.

As the appetite is usually poor and the quantity of intake insufficient, strychnin is of much value. It increases the desire for food, which, if of easily digestible nature and varied, does much to improve the consistency of the stool and the tone and contraction of the intestinal muscles. Strychnin sulphate in simple elixir is not objectionable in its taste. The usual dosage is from 1:300 to 1:100 grain, dependent upon the age and weight of the child.

Cod-liver oil and iron citrate (soluble) given in malt extract will be found beneficial where undernutrition, atony or anemia have occurred:

R	Malt extract .....	℥i	4.0
	Citrate iron (soluble) .....	grain ss.	0.032
	Emuls. ol morrhuae (50 per cent) .....	℥i	4.0
	Saccharin qs to sweeten		
	Sig. Three times daily.		

When the food intake is small because of poor appetite, it is well to use agar in the food or in an emulsion of liquid petrolatum, from 1 to 4 teaspoonfuls daily.

Agar may be incorporated in breakfast food or in biscuits, 5 grams ( $\frac{1}{6}$  ounce) of finely powdered agar to the flour necessary for each biscuit.

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## CHAPTER XIX

### INTESTINAL OBSTRUCTION AND MALFORMATION

Malformation of the intestinal tract is responsible for a small number of the deaths occurring in the newly born period and should be thought of when an infant begins to vomit soon after birth and fails to have stools containing food elements. The defect consists of an absence of a portion of the gut or of a narrowing of the lumen and may be located anywhere between the beginning of the duodenum and the lower portion of the rectum. While it is probable that most physicians have never seen such a case, at least to recognize it, the condition is being more frequently noted. Many cases, however, are not reported, so that the actual frequency is impossible to determine.

The prognosis has thus far been regarded as hopeless, and the intestinal deformity incompatible with living, but the record of at least three cases who have been saved by the operation of anastomosis makes it seem possible that in the future more of these infants will survive. In most instances the late diagnosis is the chief factor in the poor operative risk, though the shock of operation is considerable even in those cases that come to the table in good condition. There may be other factors as yet not understood which play a part in the almost unbroken series of deaths following apparently successful repair of the defect in continuity. It has been suggested by Stockard that the failure to live is dependent upon the interruption of the sympathetic nerve supply to the lower segment of the bowel whereby the rhythmic or peristaltic contractions never become developed. The most unfavorable cases are those in which the defects are multiple, or if single, the missing portion is too extensive to permit end-to-end anastomosis.

**Etiology.**—There is no adequate accepted explanation for this failure of development. The suggestion that it is due to a fetal accident does not explain the etiology, especially when more than one case occurs in the same family, in which case inheritance must play a part. Not often, however, is a second case reported in one family. Diseases during fetal life have been suggested as causative of the abnormality, but this is not regarded as probable. It is more reasonable to explain the failure to develop as due to mechanical interference with the blood supply through the mesentery whereby the portion of the intestine cannot grow. No explanation is more than theoretical, and the discussion is impractical.

**The Defect.**—The sites involved may be anywhere in the intestinal tract, but the duodenum and jejunum-ileum present the great majority of cases, with a somewhat smaller percentage in the cecum and colon. There

may be more than one intestinal defect in the same individual. A segment of the intestine is most commonly entirely missing and with it the corresponding mesentery. In complete atresia the upper and lower portions of the bowel have blind ends. Some cases have no abrupt interruption but the malformation consists of a narrow portion which is only partly patent. In others a fibrous cord connects the two ends. It is improbable that a septum within the lumen of the gut is ever present to cause obstruction. The involvement of the duodenum at the usual site of the insertion of the common bile duct is relatively frequent, in which case the bile enters either in the lower segment or above the obstruction.

Congenital obstructions of the large intestine, chiefly the colon, are much rarer than those higher up. They are more apt to be due to causes outside of the gut than to absence of the intestine. The latter, however, may be multiple, involving portions of both the small and large intestine. Absence of a portion of the ileum has been reported. The stomach becomes greatly dilated and the intestine will likewise be enlarged above the point of obstruction. Beyond the obliterated portion will be found the remainder of the intestinal tract, which may have further deformities or be patent and collapsed.

Mucous and epithelial plugs have been reported in the newly born. Cystic tumors of the intestinal wall, and hemangioma of the ileum have caused obstruction.

**Symptoms.**—The three principal symptoms which are present in all cases of complete obstruction are persistent vomiting, absence of fecal stools and the rapid or gradual appearance of abdominal distention.

One should not forget when encountering vomiting so frequently in children, that the cause may not be trivial, and that it is always present in intestinal obstruction. In the first few days of life, repeated vomiting consisting of the entire meal can scarcely mean anything else than malformation of the digestive tube. The child begins to nurse vigorously and each time the food reaches the obstructive site it is promptly regurgitated or expelled forcibly. The vomitus is bile stained if the obstruction is below the entrance of the bile duct in the duodenum. Incomplete obstruction also will cause fecal regurgitation.

The stools become smaller as the meconium disappears, and finally are absent or consist only of a small amount of mucus. Absence of bile in the meconium is shown by the light color of the passages, and indicates the location of the atresia below the papilla of the duodenum. Gas bubbles are not present unless air has been introduced by rectal injection.

Abdominal distention occurs as a result of the marked dilatation of the stomach and of the intestine as far as the site of obstruction. It is above the umbilicus, chiefly in the epigastrium, when the duodenum is involved. The lower segment of the intestine is collapsed. The infant, however, does not cry with colic or pain, except from tenderness when the abdomen is

palpated. The umbilicus may be prominent. No gurgling can be heard.

Peristalsis is visible in the region presenting distention, due to contractions in the upper segment of bowel. It may be active or feeble. Waves and patterns seen generally over the abdomen indicate an obstruction below the jejunum.

The general condition of the infant may be good until the effect of dehydration and starvation appears. Toxemia and loss of weight are then soon evident. Jaundice when present is quite definite. The baby becomes cold and somewhat cyanotic, and the temperature subnormal. The infant's facies and listless attitude show that the condition is a serious one. The urine is scanty and bile-stained, or may be absent.

The radiogram should be used in the diagnosis of congenital obstruction. The abrupt ending of the intestinal lumen is clearly shown after a barium meal.

When it is possible to secure a chemical examination of the blood, much confirmative evidence may be obtained. In high intestinal obstruction, the blood chlorids are found to be about one-third or one-half reduced in quantity. There is a marked increase in the urea and bicarbonates of the blood.

The following case of complete atresia of the small intestine is taken from the records of the Kansas University Hospital:

Female, white, aged three days, admission temperature 100° F. Since birth the child's bowels had never moved and she had frequently vomited fecal material. When brought in she was apparently suffering some pain. Examination showed a distended abdomen over which marked peristaltic movements could be seen. Palpation revealed no masses and rectal examination was negative. The abdomen was opened through a right rectus incision. As soon as the incision was made a portion of tissue resembling an appendix protruded into the incision. This proved to be a portion of the small intestine which ended blindly. About this region several inches of the small bowel was folded in an indescribable mass. There was considerable plastic peritonitis about this mass.

An enterostomy was done, the proximal portion of the intestine being brought through the wound and opened. The child did not vomit following operation. Feedings passed out through enterostomy opening in about five minutes. Patient gradually grew weaker and died in a few days.

With incomplete obstruction (stenosis) the symptoms are much the same, but death may not occur so promptly. The lumen is open, but much reduced in size. This type of congenital obstruction is illustrated in the following infant whose symptoms we thought at first were due to pyloric stenosis:

Baby M., aged two weeks, was brought into the hospital because of vomiting since the tenth day, loss of weight, and slight bleeding from the umbilicus. There was a history of blood-streaked vomitus and black tarry stools. The weight at birth was not known, but on admission it was 4 pounds, 6 ounces.



The diagnosis offered considerable difficulty: first, because the infant was ten days old before vomiting commenced; second, because the roentgen picture showed that all of the barium was out of the stomach within twelve hours and the pylorus at least not completely obstructed; third, the stools were bloody; and fourth, peristalsis was not visible. The conclusion was reached that a malformation was present in the duodenum. Thick feeding, atropin, breast milk and other dietetic remedial measures were without influence upon the vomiting. The weight, however, increased, probably from edema. The vomiting then became worse and the child died on the thirty-fifth day of age. At autopsy the stomach and most of the duodenum were found greatly dilated, but at 4 inches from the stomach, the duodenum became abruptly constricted to about the size of a lead pencil. It was, however, patent and empty. The entire small intestine was bound down in a dense mass and appeared slightly hemorrhagic. The colon was very small and contained small accumulations of inspissated feces. Apparently there had been intra-uterine inflammatory adhesions of the small intestine.

*Extrinsic Obstruction.*—Another type of obstruction is due to external causes, there being no missing portion of the intestine. In such cases there are various abnormalities found, such as incomplete rotation of the intestine resulting in volvulus, kinking of the flexure at the duodenojejunal angle as a result of traction by the mesentery or persistent peritoneal bands, pressure from congenital tumors or cysts either in the intestinal wall or adjacent thereto, also from the anterior mesenteric artery as it crosses the duodenum. The symptoms in this type are not so distinct as those found in atresia; there are usually recurrent vomiting attacks with pain in the abdomen. Symptoms may appear in infancy or not until later life. These cases are probably not so uncommon for the reason that peritoneal and mesenteric abnormalities are frequently found at autopsy. The periodic attacks most often resemble cyclic vomiting, but in infants the digestive disturbances are often wrongly regarded as simple dyspepsia. Vomiting occurs immediately after meals; the vomitus always contains bile and afterwards blood. The radiogram may be the only means of determining the site of these partially obstructed intestines. When barium is given the opaque substance traverses the duodenum slowly instead of almost instantaneously as in the normal child. Waves of peristalsis can be seen.

*Diagnosis.*—When the first portion of the duodenum is missing, the symptoms are not unlike those of pyloric stenosis. I have seen one case of jejunal obstruction which was also similar, but in these cases there is never any tumor present and the peristaltic waves are less definite.

Uncontrollable vomiting and absence of fecal stools at once indicate alimentary obstruction. In the search for the site, the beginning and end of the digestive tube should first be examined. Pyloric stenosis or duodenal atresia becomes the most probable cause of the symptoms. There will be visible peristalsis in the epigastric region in both these abnormalities. Seldom, however, is pyloric obstruction present in the first two weeks of life, and in duodenal obstruction there has previously been some food pass-

ing the pylorus. The barium meal progresses only as far as the intestinal obstruction, where the opaque substance outlines an abrupt ending of the gut.

In congenital obstruction beyond the duodenum, visible peristalsis is more apt to be found below the umbilicus, or, to be general over the abdomen. The gaseous distention is likewise more diffuse. The vomiting may be somewhat later in appearance but will soon become similar no matter where the obstruction be located. The vomitus will, however, contain more fecal material, and fecal accumulations may be felt through the abdominal wall. Weight loss and toxemia always appear. The marked reduction in the urinary and blood chlorids has been shown by Haden and Orr to occur regularly in obstruction of the upper intestine but never in the lower.

**Prognosis and Treatment.**—Death from atresia occurs within nine days, but the infant with stenosis may live a few weeks or months. While the mortality is 100 per cent in untreated obstruction, it is possible for a cure to be obtained, in some cases, by surgical intervention. This is as yet usually unsuccessful, but the technic may be developed to the point where a solution of the problem may be found. Operation introduces the element of shock and infection, and the infant thereafter seldom survives longer than twenty-four hours. The most hopeful cases are those which have a single and small defect. The function of the distal segment apparently may never develop even after apparent successful operation.

The early daily injection of physiological salt solution, in the vein or under the skin, should be practiced both before and after operation. This brings the chlorids of the blood up to normal, increases the urine, and prolongs life.

A successful case reported by Cameron from Guy's Hospital, London, was in a young infant having only one-half inch of missing intestine. The operation consisted of a posterior gastro-enterostomy.

**Volvulus.**—Volvulus is a rare occurrence in the newly born, but it may result from twisting of the mesentery, or one loop twisted around another, whereby the small intestine becomes strangulated. The symptoms are vomiting, visible peristalsis, obstipation, toxemia, cyanosis and stupor. During the stage of shock the temperature is subnormal, but becomes elevated when peritonitis develops. The upper half of the abdomen is full, the veins enlarged and the lower portion somewhat sunken. The strangulated gut becomes gangrenous, the peritoneal cavity full of fluid and death soon occurs.

**Treatment.**—At operation in the early stage the intestines should be untwisted, and fixed so that rotation cannot recur. Gangrenous involvement is usually resected, but without avail so far as life is concerned.

**Duodenal Obstruction from Peritoneal Bands.**—Incomplete obstruction or traction from bands may be present during infancy or later childhood. These bands run from the duodenum to the gall-bladder and are not uncommonly found in infants. Their clinical significance is greater than

heretofore appreciated. The congenital fixation of the duodenum causes chronic or recurrent disturbances, with acute exacerbations. The manifestations are not well defined, but they may simulate chronic colitis, intestinal intoxication, stasis or adhesions, and recurrent vomiting. Attacks of epigastric pain, digestive and toxic disturbances are the most common symptoms. Cases may live to adult life with a history since childhood of recurrent epigastric pain and vomiting.

In addition to these constricting bands there may be an absence of the muscular layer of the intestine, as a result of which peristalsis is prevented in this affected portion, partial obstruction and vomiting resulting.

The observation of two children dying with symptoms of incomplete obstruction of the duodenum was recorded by Haden and Neff. The clinical picture is that of intractable vomiting of obscure origin as the chief manifestation and acute constipation or small stools of the starvation type. The urine contains acetone bodies, and the blood shows a marked depletion of chlorids and an increase in bicarbonate of soda. The reduction in the chlorids is a finding of great diagnostic benefit. The radiogram will show that the barium passes the kinked or compressed area slowly if at all. In three cases which I have seen, death occurred, the child dying in convulsions.

A five-year-old girl without a history of previous attacks began suddenly to have pernicious vomiting and on the thirteenth day was brought to the hospital. The abdomen was sunken, but there was no evidence of usual signs of obstruction. The temperature previously normal, then became elevated. The obscurity of the origin of the vomiting led us to examine the blood to determine its chemical contents. These were found abnormal. The sodium chlorid was found to be less than half the normal (230 Mg. per 100 c.c. of blood), the nitrogenous retention (N.P.N.) was markedly increased (41.6 Mg. per 100 c.c. of blood) and the carbon dioxid combining power was high. Death occurred during a convulsion. The autopsy showed not only obstruction of the duodenum near the pylorus but also at the duodenojejunal juncture, from pressure of adhesive bands between the duodenum and gall-bladder.

Another case was that of a twelve-months-old infant, previously well, taken suddenly sick with vomiting and an elevated temperature. Convulsions occurred on the second day, the vomitus became bile-stained, the stools growing smaller and never containing blood. The abdomen was somewhat distended but never rigid nor showing peristalsis. On the sixth day of vomiting the child came under my observation at Kansas University Hospital. The blood findings were similar to those of the preceding case. The condition of the child was so low that operation was not attempted. Death occurred during convulsions. The autopsy showed a dilated stomach, an obstructive kink in the pylorus and a band of fetal adhesions extending from the duodenum to the gall-bladder. The intestines were otherwise normal. Death was due to partial pyloric and duodenal obstruction.

*Treatment.*—Persistent vomiting from this cause should be treated by an intravenous injection of physiological salt solution, followed within a few hours by laparotomy and division of the obstructing bands. In the



cases seen by the writer these methods used early would have undoubtedly saved the lives, as no cause of death other than obstruction could be found at autopsy. The condition as a clinical entity in childhood has only been recently described, but numerous recoveries by operation have been reported in adults.

**Intestinal Stone.**—Enterolith is rare in the human, but it does occur as a cause of intestinal obstruction. In 1924 Miller and Neff reported a case occurring in infancy; no other case was found reported in children under six years of age. The case seen by the writer was in a breast-fed infant who developed a total obstruction at the age of eight months. The infant had been constipated since birth. When complete obstruction occurred the abdomen became distended, fever and leukocytosis appeared, and the Roentgen picture outlined a mass in the sigmoid flexure which could be felt under relaxation from a general anesthetic. The stone was removed at laparotomy, and the infant made a normal recovery. Analysis of the stone showed it to consist mostly of calcium carbonate and phosphates, and a small amount of magnesium carbonate. When a mass is felt in the abdomen of a child having a history of protracted obstipation, a radiogram should be taken first without, and then following, a barium injection.

**Fecal Impaction.**—Fecalith or fecal impaction occurs in the intestine due to stasis of inspissated feces. Foreign bodies may become the nucleus of a mass covered with fecal accretions and coated with mineral deposits from the intestinal juices. Obstruction from such a mass rarely becomes so complete as to bring the child to operation. I knew of one case which, under the diagnosis of intestinal sarcoma, was operated upon. Ordinarily the use of laxatives and enema is sufficient to aid in the passage of the mass. One should suspect a chronic dilatation of the intestine (Hirschsprung's syndrome-megacolon), as existent when multiple or recurrent fecal impactions occur. Attention should be paid to more regular attempts at stool after such a condition has once appeared.

**Congenital Dilatation and Hypertrophy of Colon (Hirschsprung's Disease).**—The principal characteristics of congenital megacolon are:

- Congenitally enlarged abdomen*
- Obstinate constipation since birth*
- Enormous dilatation of the colon and rectum, clearly shown by the barium-injection radiogram*
- Accumulation of fecal masses felt through the abdominal wall and disappearing after thorough purgation*
- Constant or intermittent toxemia from intestinal stasis*
- Hypertrophy of the wall of the large intestine*

**Nomenclature and History.**—Congenital dilatation and hypertrophy of the colon is the term which best denotes the malformation. The condition is a deformity rather than a disease.



"Megacolon" and giant colon are terms frequently applied. About a century ago the condition began to be mentioned in the literature, and in 1870 a case with autopsy was reported by Abraham Jacobi. It was in 1886 that Hirschsprung, in Berlin, gave the description of the syndrome to which his name has since been given.

*Etiology.*—The original conception of the Hirschsprung's syndrome is that of an idiopathic malformation in which the dilatation of the colon and thickening of the walls are primarily congenital. The obstruction which occurs is of functional origin and not due to anatomic stenosis. In fact if, in the colon, there is any interference with the lumen such as stricture, kinking, twisting or peritoneal bands, the proximal dilatation which occurs cannot be classed with the Hirschsprung type, but must be regarded as secondary. Certain cases are wrongly considered as true megacolon in which during life there is a ringlike spastic contraction of the sigmoid, and an enlargement of the portion above it. Such a contraction in early life could result in hypertrophy of the colon, by interfering with bowel movements. When one exists it is located at the region of the so-called third sphincter.

This type of hypertrophy is essentially one of early life. Jacobi believed that the relatively longer colon and numerous convolutions in infancy and its greatly lengthened mesentery are the anatomical factors concerned in this maldevelopment.

The best explanation is that the malformation is congenital, and a result of primary muscular hypertrophy beginning in fetal life, and as the child grows older the intestinal wall continues to develop in thickness and circumference as a result of functional obstruction.

*Anatomy and Pathology.*—The essential anatomical change is hypertrophy and hyperplasia of the circular muscular tissue of the colon. It is thought also that the sigmoid flexure is deficient in nerve tissue. The wall of the intestine is unusually vascular. Active peristalsis is absent in the affected portion but is present in the remainder of the intestinal tract. No anatomical obstruction is found when the intestines are opened. The greatly thickened wall occurs chiefly in the neighborhood of the sigmoid flexure, but in some cases the whole colon is hypertrophied. Since the use of opaque injections and roentgenology have made it possible to visualize the large intestine, it is seen that the dilated colon occupies nearly all of the greatly enlarged abdominal cavity. Fecal accumulations may reach an enormous amount.

One of the complications occurring in these children is an injury to the intestinal wall. Deep ulcers of unexplained origin in the mucosa have been found in some cases. These may go on to perforation and peritonitis.

The greatly distended abdomen pushes the diaphragm upward and at times interferes with the respiration and probably the circulation in the chest. As a result of the absorption of toxic substances and the long-continued periods of fever, the nutrition becomes greatly impaired. In

severe cases the general muscular state is atonic, and the body growth is delayed.

*Symptoms.*—The abnormal condition can be recognized at birth in most cases, but in others the early manifestations are not evident or are overlooked.

The characteristic primary symptoms are chiefly three: great distention of the abdomen, persistent and often intractable constipation, and visible peristalsis in the lower portion of the abdomen.

The abdomen is abnormally large even from early infancy, and steadily grows more prominent. Its shape is spherical and the enlargement most noticeable above the umbilicus, probably due to the great increase in size of the transverse colon. Distention varies with the amount of fecal retention, and there is usually much gas present.

Constipation begins at birth and is constant. It is more difficult to relieve than that which is found in any other chronic obstruction occurring in childhood. For days or weeks no stools may occur. The feces become desiccated and masses may be felt through the abdominal wall. The fecal bulk becomes enormous, which can be demonstrated by the increase in the body weight and by the demonstrable loss of weight after evacuation, as well as by weighing the stools.

Many of the symptoms are secondary to the retention of feces. The disposition of the child varies with the abdominal distention. After a thorough evacuation he becomes happy and comfortable. When there has been no bowel movement for many days the child is peevish, restless, and the discomfort from distention and respiratory embarrassment is marked. Vomiting occurs during these exacerbations.



FIG. 43.—OLDER CHILD WITH TYPICAL HIRSCHSPRUNG'S ABDOMEN

The temperature will be normal except when the periods of increased intestinal toxemia develop, during which a sharp rise occurs. Fever persists until the bowel is thoroughly cleaned out. The toxemia from retained fecal material and intestinal stasis is a symptom of much importance. Its continued presence markedly affects the health and nutrition of the child, though in mild cases the growth is not impaired.

The examination of the urine is not of much diagnostic help, but it nearly always contains some indican, especially when retention of feces has been present for several days. This is significant in the breast-fed infant, who normally does not have this ammonia derivative in the urine. There are no other characteristic urinary findings.

The blood shows a progressive anemia as the cases advance in severity.

The radiogram of the abdomen is always of assistance in showing the colonic dilatation and excluding tuberculous peritonitis.

The photograph (Fig. 44) is that of a boy twenty-four months of age. His large abdomen had been noticed since he was two months old, while on

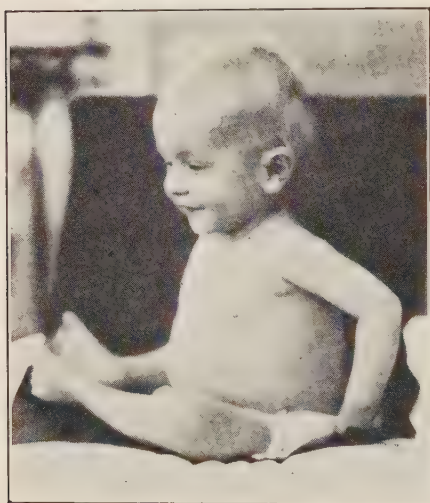


FIG. 44.—LARGE ABDOMEN DUE TO HIRSCHSPRUNG'S DISEASE

breast-feeding. He was under observation for several months at the hospital and the symptoms varied from day to day and from month to month. The size of the abdomen fluctuated somewhat, probably due to the presence or absence of large amounts of feces. Fecal impaction often caused him to weigh several pounds more than the week before. The boy often became listless and irritable when disturbed, corresponding to the time when his temperature would be above normal and in the neighborhood of 101° or 102° F. It was impossible to get him to gain in weight. He was referred to the surgeon for a first-stage operation: an ileostomy was done, an artificial anus created and the boy was irrigated through the colon each day. The later history after leaving the hospital has been that he has made considerable improvement. In late childhood it is planned to perform a

resection of the sigmoid if his symptoms have not disappeared.

*Diagnosis.*—Not all large colons date their abnormal development to the prenatal period, but may result from prolonged or recurrent fecal impaction.

Chronic intestinal indigestion (celiac disease) is most commonly mistaken for Hirschsprung's syndrome. The colon and abdomen are greatly enlarged but this condition develops late in infancy or in early childhood. The stools are bulky, usually numerous and diarrheal. When constipation exists the diagnosis between celiac disease and megacolon is not easy, and



must be made from the history and the composition of the stools, which are entirely different in the two conditions.

One should not place too much importance upon the finding of a dilated colon when the typical history and the other symptoms are absent. Not infrequently the colonic shadow may be greatly enlarged at one roentgen examination and found to be down to normal at another time. On the other hand, it is easy to understand why the diagnosis of megacolon is so often erroneously made by clinical observation alone, for there are many border line conditions which cannot be excluded except at autopsy or operation. The history, since birth, of dilatation and constipation and the thickening of the intestinal wall are necessary for the proof of Hirschsprung's syndrome. Hypertrophy of the wall can only become proven when the abdomen is opened.

Obstinate constipation may be due to visceroptosis, intestinal redundancies, loops, kinks, dilatation from anatomical obstructions. It is probable that Hirschsprung's syndrome, which is rare, is often diagnosed when these conditions are present.

The pot-belly and constipation of rickets are not congenital, though they may begin in the first four to eight months of infancy. Other symptoms are evident, but there is no reason why both rickets and Hirschsprung's syndrome might not coexist.

Sarcoma of the intestine was the preoperative diagnosis in a child whose previous history was not carefully obtained. The abdomen was large, a tumor was clearly palpable, but at operation the mass proved to be a fecal impaction, and the colon was found to be a typical Hirschsprung's deformity.

The second most common error in diagnosis occurs with tuberculous peritonitis. This disease is common and is better known. The abdominal enlargement is recent and more rapid. Fluid is usually demonstrable. A positive tuberculin test is obtainable, and it may be possible to find a tuberculous history and physical evidences elsewhere in the body.

*Medicinal Treatment.*—Satisfactory results in mild cases are obtained from the use of enemata and laxatives. Castor oil should be an ingredient of both of these. Removal of the fecal masses which are felt as tumors is accomplished by the daily administration of oil by the mouth. A few hours later a high large injection of soapsuds and oil is given. A protracted period of such active treatment should undoubtedly precede any operative interference, as children become weakened and anemic from a long-existent toxemia from retained feces. Many drugs have been tried, such as epinephrin, pilocarpin, physostigmin and atropin. The latter drug, in doses of 1:500 to 1:100, will benefit those atypical cases in which enterospasm may play a part. A routine daily laxative of merit is the mixture of aromatic cascara and fluid extract of senna, in doses of a teaspoonful or more.



*Dietary Treatment.*—During periods when fecal impaction exists the diet should be largely liquid, so as to add little residue to the intestinal contents.

Constant study should be made of the diet which best suits the individual child. For the infant, breast milk in large quantities is usually productive of soft stools. This may be supplemented by laxative fruit juices, vegetable soup and olive oil. Coarse fibrous foods with much residue are here of less value than in simple constipation. Artificially fed infants should have a low milk and high carbohydrate formula which prevents the hard calcium stool. In spite of dietary treatment, constipation is usually persistent and obstinate. It can be said, however, that diet is an adjuvant to the general amelioration of this condition.

In older children, in addition to attempts in finding a laxative diet, there should be:

*Hygienic Measures.*—Massage of the abdomen, regular hours for going to stool, deep breathing, exercises for developing the abdominal muscles and assisting peristalsis should be begun as soon as the child is old enough to assist.

*Surgical Treatment.*—The danger from operation is in the resulting shock and peritonitis. The probability of peritonitis is lessened when the large intestine is previously opened (appendicostomy or colostomy) and well drained. Mobilization of the large intestine helps to prevent peritonitis. The establishment of a fistulous opening enables the intestine to be kept well drained, and the child to recover from the chronic toxemia.

The second stage of the operative treatment is the excision of the terminal portion of the colon. The first part of the gut should be spared if possible, for it takes part in the physiology of sugar and albuminose absorption. Sistrunk has advocated the radical resection of the sigmoid portion of the colon after a careful first-stage drainage operation; the resulting mortality in recent years with the improved method is as low as 5 per cent.

*Summarizing* the handling of congenital dilatation and hypertrophy of the colon, I would emphasize the importance of a careful history and examination; the treatment by diet, laxatives and hygienic care; prevention of long-continued toxemia; the general health of the child to be improved by outdoor life and the administration of iron-containing foods. If the welfare of the child permits, there should be no operative interference in the tender years of life, a preliminary operation should be done for direct drainage by the creation of a fistula which is to be kept open, and the child's health brought to the normal. Finally radical operation may be necessary by removal of distal portion of the colon.

*Course and Prognosis.*—Occasionally mild cases recover without surgical treatment. The rule is for the gradual failure of the health, hemorrhage from the intestine, perforation and peritonitis, respiratory disease and death. Hirschsprung's syndrome may cause a fatal termination in early infancy,

recovery may take place in other cases, but chronic invalidism is the most usual course. If the child can be maintained in nutrition until adult growth is reached, the outlook is then favorable.

Surgical treatment has been unavailing until recent years; the methods recently employed offer much promise of curing the condition and saving life.

**Congenital Microcolon (Small Colon).**—A rare form of congenital intestinal obstruction, but which produces prompt symptoms after birth, is a congenital narrowing of the canal of the colon, associated with a thickening of the wall. The hypertrophy of the wall is similar to that of Hirschsprung's deformity, but instead of dilatation, the lumen is abnormally small, though open. However, it produces a complete obstruction. The meconium collects in the dilated ileum and cecum, becomes thickened, and does not pass into the colon.

Symptoms are those of idiopathic obstruction consisting of persistent vomiting, absence of stools, rapid emaciation of the body and the absence of peristalsis in the colon. The abdomen becomes distended. Masses can be felt through the abdominal wall, but no tumor can be palpated per rectum.

At operation or autopsy the colon is found to be a small firm tube, the coats of the gut perfectly formed, the mucous layer containing glands, but the general function defective. According to Grieg, microcolon is a physiological abnormality, with no anatomical defect other than the small size. The condition is incompatible with life.

The diagnosis from other low intestinal obstructions is impossible, unless barium injected into the rectum can be made to reach the lumen of the colon, where it will be seen as a narrow opaque line. From the fact that no meconium stools have occurred, one can infer that a low obstruction is present. Atresia or absence of segments of the colon produce the same symptoms and clinical picture.

*Treatment.*—Operation is indicated at the earliest moment. A short-circuiting of the functioning intestine above and below the obstruction is necessary. Thus far all reported cases have died.

**Disturbances of Meckel's Diverticulum.**—Intestinal obstruction and diverticulitis occur as the most frequent complication from the presence of a Meckel's diverticulum in an individual. It should be remembered that a perforation of the diverticulum may come from the occurrence of a peptic ulcer of this stricture.

*Anatomy.*—This diverticulum is a pouch originating in the ileum close to the cecum. It persists in many individuals as a remnant of the embryonic duct connecting the yolk-sac with the intestine (omphalomesenteric duct). Numerous forms are found. The open form communicating with the umbilicus is of chief interest because of its manifestation at the surface of the body. Its presence is recognized in the newly born infant when the cord comes off. An open diverticulum reveals itself in a discharging fistula at the navel. A diagnostic point is the discharge of an alkaline secretion.

The type communicating with the umbilicus also shows sooner or later an escape of gas or small amounts of feces. Protruding from the mass will be the reddish mucous membrane of the intestine. It should be thought of in atypical cases of umbilical hernia, granuloma or urachal fistula. The tendency and menace of this type of diverticulum are the production of prolapse and obstruction of the intestine.

The usual or blind type is seen only at operation or autopsy. The diverticulum ends in a cordlike appendix or ligament. This may be free in the abdominal cavity, in the mesentery, or may be found in the sac of a hernia. The cord may be attached to the umbilicus. An illustration of this type occurred in a recent case at the Kansas University Hospital:

A white female infant died at one month from weakness due to prematurity and congenital heart disease. She had numerous malformations of the heart, a club hand, a horseshoe kidney and a Meckel's diverticulum which existed as a solid cord leading from the bowel to the umbilicus.

*Volvulus*.—A case of acute intestinal obstruction of five days' duration, brought into the University of Kansas Hospital, was of extreme interest because of its rarity.

This two-year-old child had, in addition to constant vomiting, an absence of stools, no hemorrhage, but the intestinal patterns were seen over the entire abdomen. There was no abdominal tumor. The child had low blood chlorids and carbon dioxid combining power, the latter being evidence of marked acidosis. At operation, the large intestine was found dark in color and the ileum showing a volvulus caused by a Meckel's diverticulum which was about 2 inches in length with the distal end free in the peritoneal cavity. The child was given sodium chlorid solution under the skin and glucose solution by the vein, but died twelve hours after the operation.

*Ulceration*.—Ulcer may occur in the diverticulum or near by in the ileum. It is essentially a disease of childhood. While I have been able to enumerate at most only eighteen cases in reports from the literature, the finding of this condition at operation and the frequency of Meckel's diverticulum make it probable that the disease is more common than reports indicate. Among these, two were in infants with symptoms beginning before five months, one at eleven months, four in children from four to five years, seven between nine and eleven years. Thus far only boys seem to be affected.

The pathological anatomy is concerned with the anomalous existence in the diverticulum of gastric mucosa with acid-secreting glands, and the glands of Brunner. This histological misplacement is found in 12 per cent of diverticula. The intestinal mucosa is also present in the diverticulum. The ulcers develop at the edge of the zone of this misplaced gastric mucosa, and resemble the gastric or duodenal type of peptic ulcer. The ulceration may readily reach the serous layer of the diverticulum and perforate. It



results from the acid or peptic secretion from the misplaced gastric cells attacking the epithelium of the alkali-secreting portion of the diverticulum. Just why ulcers occur in one diverticulum and not in another, when so many have the gastric cells present, is not to be explained, but probably due to the same factors concerned in the causation of gastric and duodenal ulceration. Strauss reported one case in which ulcer occurred in the dome of the diverticulum.

Peritonitis and death result from perforation if not surgically treated.

*Symptoms.*—The first symptom is the copious intestinal hemorrhage. The child may have been previously either well, or have complained for sometime of abdominal pain. Bleeding from the bowels occurs several

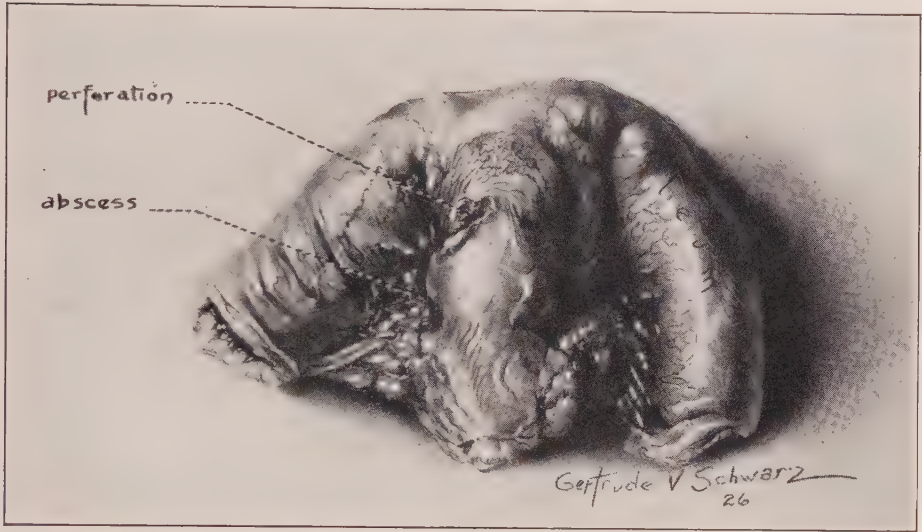


FIG. 45.—PERFORATION OF MECKEL'S DIVERTICULUM

This occurred in a ten-year-old boy. There was also present an adjacent abscess.  
(Courtesy of L. F. Barney, University of Kansas Medical School.)

times daily and consists of dark-red blood, not changed by digestion. It may amount to several ounces and continue for days or weeks with the production of severe anemia and even death. If the child lives and does not undergo perforation of the ulcer, the hemorrhage abates for awhile and returns in a few weeks or more. The tendency to recurrence of a copious, red, hemorrhagic stool is diagnostic.

Perforation is the most common complication and was present in nearly all of the reported cases. It may open into the peritoneal cavity, mesentery or abdominal wall, with the development of adhesions and localized abscess. The perforation may appear within a week or not till several months after the last hemorrhage. Abdominal pain may precede the perforation, but it is indefinite or resembles colic. The young child is incapable of giving a clue to the nature and location of the pain. Palpation reveals no distention or tenderness.



A case which I saw in the surgical practice of Edwin Lee Miller presented acute pain and symptoms referable to the bladder:

An eleven-year-old boy began to have abdominal pain, hemorrhagic stools, and irritation of the bladder. There was a constant desire to urinate not relieved by voiding. This was due to the proximity of the bladder to the diseased area. Six hours later symptoms of shock and diffuse peritonitis were present. The white blood count was 25,000. Laparotomy revealed a perforating ulcer of Meckel's diverticulum, the unaffected areas appearing of the same color and character as the other portions of the small intestine. The treatment consisted of resection of the diverticulum, intestinal suture and drainage of the peritoneal cavity. Recovery occurred.

The disease may last for years before perforation occurs, beginning in early childhood and lasting till early youth. But it is the tendency to this serious complication that is dangerous.

The differential diagnosis is to be made especially from intussusception. Invagination is found in infancy and is far more common. Vomiting and colic are likewise more frequent and severe. The hemorrhage is less in amount and the blood is mixed with mucus. Tumor is more apt to be present, but peritonitis may result in both diseases. The absence of pus in the stools rules out dysentery. So-called melena occurs in the early days of life. Peritonitis brings into question the diagnosis of appendicitis, which is usually first considered, and for which the case is operated upon. The clinical picture is not well known unless the surgeon has previously seen a similar case.

*Treatment for Ulcer.*—Removal of an ulcer before perforation occurs is the ideal but is seldom done because of the diagnostic difficulty. When bloody stools have developed the immediate surgical treatment is indicated. Seven of the reported cases have been saved by operation.

*Treatment for Intestinal Obstruction.*—In the umbilical type, prolapse of the diverticulum and resulting obstruction calls for immediate operation. Otherwise operation may be postponed until later infancy.

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## CHAPTER XX

### ACUTE SURGICAL DISEASES OF THE INTESTINES

#### APPENDICITIS IN CHILDHOOD

**Descriptive Summary.**—Acute appendicitis in childhood is to be regarded as a strictly “surgical disease,” beginning in the appendix and extending rapidly to the peritoneum. Pain, crying and in older children abdominal tenderness are the only subjective symptoms of importance. The objective symptoms of diagnostic importance are localized rigidity of the right abdominal muscles, becoming diffuse over the abdomen in the course of twelve to forty-eight hours; the vomiting is early, and may be continuous; there is an increase in the white blood-cells of the blood, chiefly in the lymphocytes on the first day and afterwards in the polymorphonuclears; a tumor mass is felt through the abdominal wall or per rectum. While the disease is necessarily one for immediate surgical intervention the physician, as first on the ground, should be able to recognize promptly its diagnostic features and call the surgeon at once. He should see that no sort of laxative is given, even though not all of the typical symptoms are found.

**Nomenclature and History.**—Before the nature of the disease was understood, the cecum was supposed to be the viscus affected, and the term typhlitis was used. This is now known to be incorrect, as the appendix and not the cecum is concerned in this infection. Therefore the name appendicitis has entirely supplanted all others.

Little was known about appendicitis in childhood until the advent of modern surgery, better methods of diagnosis, and autopsy examination, and until the development of current literature revealed the fact that children have the disease not uncommonly. Surgeons are now reporting series in childhood, recognizing that the course and treatment require different consideration at this age.

**Etiology.**—*Age.*—The incidence of the disease in childhood shows that age is a factor in the etiology. The disease becomes more frequent with each year of life. One case of fetal appendicitis has recently been reported, and a few have been encountered in the newly born period (two weeks) and during the first year. While not common under three years of age it should be remembered that no age is exempt. Abt, in 1917, reviewed eighty cases which had been reported under two years of life. Most reports, however, deal with the isolated case. Bolling, in a symposium before the Children's Section in the American Medical Association, 1924, reported 123 personal

cases in children, forty-two of whom were under six years of age, the mortality proving higher in the younger children. When all ages are considered, the incidence in childhood is found to be 3.5 per cent in the first five years, 8.5 per cent in the second five-year period, and 16 per cent in the third.

*Sex.*—It is generally recognized that this disease is more frequent in males. Brown, in the study of seven hundred cases, found that there were twice as many boys.

*Microorganisms.*—Appendicitis is not caused by any one type of organism. The colon bacillus, *Streptococcus hæmolyticus*, *Proteus vulgaris*, *B. pyocyaneus*, the pneumococcus, staphylococcus, and others less commonly have been reported. The infection is carried from foci of infection elsewhere, especially from the throat, and in the case of the colon bacillus from intestinal infections. The infection is hematogenous in most instances, rarely local through the mucosa of the appendix. The appendix may be involved in typhoid fever as a rare complication. Diet as a cause in children is not a factor.

There is much reason for concluding that pin-worms (oxyuris) and possibly other parasites may cause appendicitis. For three centuries it has been known that pin-worms may infest the appendix. The relation between the parasites and the disease of the appendix was emphasized by Metchnikoff in 1901. Their presence in large numbers may cause degenerative and exudative changes in the appendiceal structures. Some authorities believe that the appendix is a breeding place for the oxyuris. Harris and Browne found pin-worms in 18 per cent of 121 routine appendices examined in the laboratory. Three of these were in children. Whether or not this is a common cause, the known presence of a pin-worm infestation should suggest the invasion of the appendix when symptoms not entirely typical of appendicitis are found in a child. Such symptoms are not so severe or acute, the abdomen not so rigid and distended, the temperature normal, but there may be discomfort and pain in the region of the umbilicus or appendix and a feeling of heaviness or tightness in the iliac fossa. The attacks are recurrent, and last through a period of a year or more, with little fever or change in the leukocyte count.

*Anatomy and Pathology.*—The normal appendix at birth varies in length from 2 to 8 centimeters, and at the end of the first year reaches a total of 10 centimeters, increasing slowly thereafter. The ratio of the length of the appendix to that of the large intestine is similar in childhood and adult life. The diameter, while relatively large, is easily recognized as belonging to childhood. It is from 0.2 to 0.6 centimeter in the newly born. Its opening into the intestine through which fecal or foreign matter more easily enters or leaves its lumen, is also relatively wider than in the adult. Peristalsis is a normal function of the appendix, assisting in its emptying,

and is the probable cause of some of the pain in the early stage of inflammation. The appendix differs little in position in the child, with the exception that it is somewhat higher in relation to the plane of the pelvis which is not relatively so deep in young children. It may point in any direction. The landmarks and the varieties of direction after it leaves the cecum are identical with those found in later life. It lies to the left of the cecum more often than behind. Lymph-nodes are present in the appendix at birth, and abundant lymphoid tissue is found in the appendix of children from the second year and afterward. The younger the child the smaller the omentum, and the less the protective action toward walling off infective and suppurative processes.

The nature of the pathological involvement of the appendix itself is similar to that in adults, but in young children infection becomes widespread and does not usually become walled off by localized abscess formation. Peritonitis extends to the whole abdomen and pus may gravitate to the flank or to the pelvic region. In forty-three cases under five years of age reported by Beekman, there were only three instances of unruptured appendix, there was a localized abscess in seventeen and diffuse peritonitis in twenty-three. Inflammatory changes characterize the earliest stages of the appendiceal involvement, accompanied or soon followed by microscopic evidence of suppurative process in the structure of the appendix. The tip may be involved alone in the early stage or the entire organ may be affected. Edema, redness and hardness are evidences of beginning inflammation, which is especially noticeable in the inner coats. Necrosis and edema soon involve the whole structure and the peritoneal layer becomes covered with exudate. Thrombosis of the blood-vessels and gangrene of the appendix follow. The infection spreads to the peritoneum as soon as rupture occurs. The presence of foreign bodies in the appendix is frequently noted, but is not always of pathologic significance. Pressure necrosis from fecal concretions, foreign bodies, and interference with the integrity of the mucosa from the presence of pin-worms are of undoubted influence in lowering the local resistance to infection.

**Symptoms.**—The age and disposition of the child have much to do with the examination. In most children under five years, and with any child who is frightened, the elicitation of either the subjective or some of the objective symptoms is a matter of marked difficulty. Gentleness and kindness must be used in the approach of the examiner. Abruptness and roughness will not be the manner of the physician who is experienced in the handling of children. However, there are children who will not easily permit any attempt at a satisfactory examination. In any case it is well to elicit first the subjective symptoms and then make as complete an examination as possible. When it is impossible so to examine the child as to determine the presence of localized tenderness, muscular rigidity or tumor either



through the abdominal wall or by rectum, the physician must use his judgment as to the advisability of a general anesthetic for completing the examination.

*Children under Five Years of Age.*—In infancy and early childhood the occurrence of vomiting, severe crying indicative of pain, and evidences of tenderness on palpation are suggestive and valuable symptoms, though not specific. Pain, nausea and vomiting are present in over 90 per cent of cases. Pain is due to tension in the appendix or to its peristaltic movement. Children of this age cannot correctly indicate the presence of tenderness except by flinching, and frequently complain when pressure is made over any portion of the body. Especially after the age of one year, the increasing frequency of appendicitis must be borne in mind.

The history usually indicates that the child has been previously well. After the initial crying has subsided the child may be comfortable but prostration is evident. He does not sit up, but lies motionless, with the legs drawn up. Recurring pain may cause the child to cry constantly, scream and to be unable to sleep. It sometimes happens that appendicitis develops in infants with pain as the only early symptom. The predominant thoracic breathing which is uncommon in the normal child confirms the presence of spasm in the abdominal muscles. It may be possible to determine rigidity of the right-sided muscles, and if peritonitis is present the general abdominal rigidity.

The presence of tumor is felt with difficulty because of the general rigidity and peritonitis. Rectal examination for tumor should never be omitted.

Schwartz reported a mild and insidious case in a six-month-old infant that had a leukocyte count of 6,000 to 8,800, the temperature never above 100.2° F. and little peritoneal fluid, no vomiting, and no pain after the second day. The unusual symptom was the passage of blood and mucus.

*Symptoms in Older Children.*—From the foregoing remarks about the peculiar difficulties in determining the symptoms in early life, we can now pass to the symptomatology which is general to childhood.

*Pain.*—This is usually the first symptom. In children who have the ability to localize, it is at first usually referred to the region of the umbilicus, to appear later at McBurney's point or in the right lumbar region. Pain and tenderness on pressure will also be usually elicited at this point. Palpation is of more value than trying to determine the location of spontaneous pain. Secondary pain arises from the spread of the inflammatory process to the surrounding parts. Immediately after perforation there is freedom from suffering, until the development of generalized peritonitis.

*Vomiting* nearly always appears early as the second symptom. It may be continuous, or may disappear.

*Muscle rigidity* of the right side is of early value, but in children it

soon becomes more general over the entire abdomen. Rigidity is a reflex and protective mechanism and is present in practically every case in childhood.

Irritability of the bladder is present in some cases, due to the proximity of the inflammatory mass in the right lower quadrant.

*Tumor* is a relatively late manifestation and can be felt when the abdominal distention is not too great. When it is not recognizable through the abdominal wall, it should be sought by rectal palpation.

The *temperature* is from slightly above normal to 103° F. It is well to emphasize here that the determination of body temperature is reliable only when taken by the rectum, and that no deduction should be made because of this method of taking it. Because appendicitis can exist without marked elevation of temperature, the value of its determination as a symptom is limited.

The *white blood count* is of great aid in diagnosis but of little in determining the severity or prognosis. A rising white count from day to day is the rule. The physician should not wait for repeated blood counts in children, as it adds to the operative delay. Leukocyte counts above 12,000 and averaging 17,000 with a preponderance of the polymorphonuclears furnish a symptom of marked value in the child who has been previously well.

Constipation is usual, though diarrhea occasionally is present. The condition of the stools is not therefore characteristic.

The urine may contain albumin and casts, but shows nothing characteristic for the disease.

*Roentgen Ray*.—Nothing is to be gained by the roentgen-ray examination in appendicitis. The delay and additional handling caused therefrom is not warranted. In the inflamed appendix there would be a complete defect in filling with barium, and after perforation some barium might be found in the peritoneal cavity. The normal appendix may be clearly outlined, or it may be hidden by the intestinal shadow.

**Differential Diagnosis.**—Practically all symptoms of appendicitis may be found in other diseases of childhood.

*Lobar Pneumonia*.—Of first importance is lobar pneumonia, with its toxic distention and pain in the abdomen. The mistake is sometimes made of opening the abdomen. Shortly afterwards a physical examination of the chest shows unmistakable signs of pneumonia. While pneumonia is often incorrectly diagnosed as appendicitis, it is rare for appendicitis to be diagnosed as pneumonia. It is wise, however, to consider carefully the abdominal signs in pneumonia as worthy of attention, for appendicitis might coexist.

All cases should have a general examination, especially of the chest, and in the absence of chest signs the doubtful case of appendicitis should have a radiogram of the lungs.

DIFFERENTIAL DIAGNOSIS OF APPENDICITIS  
AND PNEUMONIA

<i>Appendicitis</i>	<i>Pneumonia</i>
Fever gradual, may not be above 100° F.	Fever sudden, 103°-105° F. and may drop by crisis
Seldom has a real chill	Chill of marked character
Pallor of skin	Flushing of skin
Normal respiratory rate	Increased respiratory rate
Thoracic breathing so as to immobilize abdomen	Abdominal breathing
Quiet	Irritability
Stupor and delirium absent	Often stuporous and delirious
Pain, tenderness and rigidity in abdomen, pain elsewhere due to fright	These symptoms are present in certain cases, especially basilar pneumonia with pleurisy. Child not frightened by fear of pain elsewhere on examination
Dulness and a mass in right iliac fossa	No localized abdominal tumor
No physical findings in chest, though the possibility of simultaneous occurrence of appendicitis and pneumonia	Definite physical signs in chest and cough
Radiogram of chest negative	Consolidated area in lung or impaired visibility
Leukocyte count: usually under 20,000	Usually over 20,000 and increasing
Urine usually normal	Pus, casts, reduced chlorids

**Other Infections.**—Upper respiratory infections such as influenza may cause abdominal pain, usually indefinite in location. Pyelitis, especially in girls, occurs with tenderness, which if in the right kidney may suggest appendicitis. The examination of the urine for pus should be a routine. Acute digestive upsets with vomiting, intestinal intoxication, and intestinal obstruction such as intussusception, are always to be considered. Fecal impaction is occasionally confusing, while early Pott's disease and diverticulitis are conditions which may come into question. Abdominal (Henoch's) purpura has been occasionally diagnosed as appendicitis.

Mesenteric lymphadenitis of the small intestine, due to tuberculous infection with pea-sized nodules in the mesentery, is not infrequent in children, and causes symptoms resembling appendicitis. These are subacute pain, tenderness, rigid abdomen in the lower right quadrant, mild fever, malaise, weight loss, and symptoms of indigestion. The condition is not usually recognized even at operation, but has a favorable prognosis.

Pain simulating appendicitis occurs occasionally during scarlet fever, and I have known such pain to follow the administration of typhoid vaccine.

It is wise to bear appendicitis in mind as the most serious cause for acute abdominal pain, and to lose no time in deciding as to its presence.

**Complications.**—Postoperative ileus is rare in childhood according to Bolling. Abscess in older children is common and diffuse peritonitis the

rule in young children and may occur in later childhood. Drainage of the peritoneum, and if the appendix be not at once removed a subsequent appendectomy, make the convalescence somewhat tedious.

**Treatment.**—Appendicitis in childhood has no medical treatment except for rest and relief of pain. Laxatives are forbidden because of their peristaltic stimulation. It is better for all concerned if the possibility of appendicitis is discussed with the parents, and the child removed at once to the hospital where the assistance of the surgical consultant and the laboratory may be obtained.

The physician thus does his duty by the child, and the family appreciates his ability in promptly recognizing the gravity of the case. The child should be operated upon at the earliest moment following the diagnosis. The details of operation are left for consideration in another volume.

As a prophylactic for parasitic appendicitis, the removal of worms from the intestinal tract is advisable.

**Prognosis.**—In the first five years of life the mortality is higher than in the remaining years. The younger the child the worse the prognosis. At this age the appendix is ruptured earlier, and usually by the time of operation. Diffuse peritonitis is the rule in young children with less tendency to protective localization. It is made more certain, and occurs at an earlier time in cases that have been given castor oil or other laxatives. There is no object in giving an enema. Certainly laxatives increase the mortality rate in appendicitis.

Mortality in appendicitis has been lowered in recent years. Most surgeons of experience in children operate upon the child when first seen, as offering the most hope of saving life.

## INTUSSUSCEPTION

*(Invagination, Acute Type)*

**Descriptive Summary.**—This variety of intestinal obstruction is found usually in previously healthy breast babies and it demands immediate diagnosis and surgical intervention. The type most commonly known is acute, but subacute and chronic cases are not unheard of. Vomiting, severe pain and screaming soon followed by a markedly hemorrhagic stool make up the early diagnostic picture. Palpation of the abdomen may reveal a mass commonly in the right lower quadrant, later anywhere in the abdomen. In from twelve to forty-eight hours a tumor may be felt by rectum. The condition is complicated at this time by distention of the abdomen and the appearance of grave symptoms such as prostration and shock. The important features of the handling of the case are the diagnosis and surgical intervention within the first few hours after the onset when the chances for life are good. The physician is urged to regard all acute vomiting with bloody stools in the nursing as probably due to obstruction, and to remember that waiting for



the appearance of an abdominal tumor jeopardizes the chances of recovery.

**Etiology.**—The obstruction occurs more commonly in males. Nearly all cases are found within the range of three to fifteen months of age. The fifty children reported by Thompson represent the common age, 62 per cent were between four and eight months of age and 88 per cent in the first twelve months.

Many theories have been advanced as to the causes. Undoubtedly most cases begin without any previous disturbance. Some have followed a digestive upset. In England and Denmark the frequent use of large doses of castor oil is said to be regarded as having an exciting influence. Excessive or irregular peristalsis produced by such causes may be important.

In the perfectly healthy breast infant some congenital developmental predisposition must be present. In such instances there is a "floating" cecum or an abnormally mobile colon, which is characterized by a long mesentery. Floating cecum is found much more commonly in infants than in older children. It is only by such abnormal mobility that the ileum, cecum and upper portion of the colon can become invaginated to the extent of reaching the last portion of the colon. It is probable that these subjects belong to the type of children who later have visceroptosis, and whose parents are of the "lanky" build. The development of the apex of the tumor is most commonly at the junction of the small and the large intestine (ileocecal). The smaller lumen of the former permits its easy entrance into the latter. Invagination can occur anywhere in the intestinal tract. Although in the great majority of cases at operation no explanation of the invagination is obvious, intrinsic factors can often be demonstrated, such as changes in the mucosa, swelling of Peyer's patches, tumors of the connective tissue in the intestinal wall, foreign bodies, ulcer and trauma. Cases have occasionally been found in infants having a congenital constriction of the small intestine. In experimental intestinal obstruction, produced by the ligation of the intestine in dogs, Orr noticed that invagination sometimes followed. Thompson found a high percentage of enlarged mesenteric lymph-nodes at operation for intussusception and he thinks that they have some significance in the etiology. It is possible that enlarged glands may affect the position of the intestine by traction or pressure. The enlargement of these glands may be due to hyperplasia or in some instances to a preceding enteritis.

Meckel's diverticulum is said to be present in 2 per cent of all individuals. It is responsible for some cases of enteric intussusception. Twelve cases of congenital mucous cyst causing intussusception have been collected by Bazin. He reported individual cases in which the cyst was in the wall of the cecum near the valve.

**Anatomy and Pathology.**—When the invagination reaches only to the ileum, it is called ileo-ileal (ileal meaning enteric). The ileocecal type comprises about 60 per cent of all cases. When the cecum is less mobile, it maintains its normal place, while the ileum slides down into the colon, form-

ing the ileocolic type. According to Thompson, 40 per cent of cases have two invaginations, a higher one in the small intestine being superseded by another which starts with its apex in the ileocecal valve. The colon and cecum form the outer and middle coats of the tumor mass, while the ileum is the inner layer. In this type much of the length of the intestine is involved. The ileocecal valve may traverse the whole length of the large intestine and has been known to protrude through the anus. A true colonic invagination sometimes occurs, an upper portion of the colon becoming invaginated into a lower. The movement of the intussusception is nearly always downward, but a small percentage of cases are of the ascending or reverse type.

In the mechanism of intussusception there is said to be at first a circular contraction of the intestine which permits of a subsequent overlapping. Experimentally the condition has been produced by tying off small segments of the mesenteric circulation. This produces a thrombosis of the vessels, a phenomenon which is sometimes observed in the clinical case.

The invaginating process is arrested when paralysis of the bowel occurs. The mesentery is drawn into the invaginated bowel and becomes constricted, shutting off the blood supply. Gangrene soon supervenes.

Simple invagination of the bowel, usually the jejunum, is often found at autopsy. It is entirely different from the inflammatory type under discussion, and is purely a terminal or agonal manifestation.

**Symptoms.**—The history of the onset is of the utmost importance. The striking feature is that of sudden, repeated and severe crying and screaming in a previously normal infant, usually breast-fed. Thompson found that this severe periodic abdominal pain with associated screaming is so typical as to be present in all cases. Crying is accompanied by drawing up of the legs. In the first twelve to twenty-four hours the child may look entirely well between the attacks of pain.

There is no fever at first, but soon an elevation occurs ranging from a moderate amount to a hyperpyrexia.

Vomiting occurs early and this may be only once and of short duration or it may be persistent. Within a few hours the child passes bright red blood which varies in amount from a few teaspoonfuls to a considerably larger quantity. As paroxysms of colic recur, more blood is passed; there is also some mucus. Bleeding is found in all of the cases except in the type which remains above the ileocecal valve, where it may be absent.

The contour of the abdomen is significant. It presents a somewhat swollen appearance. When the cecum is mobile it is carried along into the advancing invagination, past the right iliac fossa. This produces the characteristic emptiness of that region.

In emaciated children with poor circulation, intussusception may have atypical symptoms, especially a reduction in the amount of pain and evidence of distention. The peritoneum does not show the marked reaction

found in vigorous individuals, the symptoms of pain, vomiting and rigidity being diminished in poorly nourished infants and children under three years of age.

Intussusception in the dying individual may be multiple and is non-inflammatory and easily reduced.

Under very favorable conditions the tumor, which is always present, may be palpated. This mass may be found in any portion of the abdomen, since there is no part of the intestine which may not be invaded. If the infant be crying or frightened, the abdominal wall is held so rigid that one cannot feel the tumor. It is recommended that light anesthesia be administered in such instances, just enough to reduce the abdominal resistance without interfering with the intestinal spasm. Within the first twelve hours, the tumor may be felt in the right lower quadrant, following which it may be found under the right costal margin, where it is somewhat inaccessible to palpation. The mass continues to progress along the course of the intestines and may next be found above the umbilicus and later in the left upper quadrant. At the end of thirty-six hours it is hard to find by abdominal palpation. The tumor may be sausage-shaped or may vary in size and contour. It is usually somewhat movable. Rectal examination is the most important of all the later procedures, but the diagnosis is usually possible before a rectal mass is discoverable, which is usually after forty-eight hours.

A radiographic examination is of no practical help, and one is not warranted in further delay. The colonic injection of barium may reach the intestinal obstruction and the picture will show a cervix-like projection of the invaginated bowel into the opaque substance below. This gives some idea of the extent of the intussusception.

The general appearance of the infant soon changes. He becomes abnormally quiet and may be insensible to surroundings. The eyes are sunken and the child shows some shock. The amount of the shock varies, and has no relation to the extent of the intussusception. The abdomen becomes distended, which may result simply from the toxemia. Finally the bowels become paralyzed and gangrene of the intestinal wall soon follows the obstruction to the circulation. The condition of the infant grows rapidly worse and death occurs in less than five days.

**Course.**—Intussusception as it is generally known is of the *acute type*. The duration is seldom longer than three or four days. The outcome is invariably fatal in the untreated case. Each hour that the obstruction remains unreduced sees the condition of the child rapidly failing, and the chance for operative relief greatly reduced.

*Subacute* intussusception is of unusual occurrence. Its etiology and pathology are similar to those of the acute type, but the course is longer, the child living from one to two weeks. The symptoms are much less striking. The vomiting is not continuous, the pain is less severe, and the general condition not so grave. The stools are loose, watery, mucous, at the



onset always bloody, and various amounts of blood may be passed during the course of the disease. Because of the mildness of the symptoms and the absence of definite signs of obstruction, the presence of a tumor may be overlooked. The disease resembles dysentery and renders the diagnosis difficult. In the cases reported by Bergstrom one infant showed little disturbance at any time, while another developed coma. Two infants sick eight days recovered by operation, and one sick sixteen days died. The obstruction in all cases was incomplete and as gangrene had not developed resection of the intestine was not performed. The fourth case was in an eight-year-old boy sick eight days, without blood in the stools, the diagnosis being made by the recognition of the tumor in the descending colon.

*Chronic* intussusception is of rare occurrence, but one can find about twelve cases thus far reported in children. The chronic type really belongs to adult life and is usually secondary to a new growth. In childhood it occurs after the age of one year. Still has seen four cases, three of whom were in the second year. Marsh described two cases in boys of two and one-half and three years of age, while an eight-year-old girl is reported by Yamagiski.

The duration from the beginning of symptoms until the disease is diagnosed and operated upon has varied from weeks to months. Just how much longer the child might live unassisted is undetermined. Because of the mild symptoms and the peevishness of the child, the condition may be ascribed to so-called "biliousness." There is no suspicion of the true nature of the disease. The bowels are open though somewhat constipated and at no time is there complete obstruction. There is usually no blood in the stools at any time. One observer mentions the occurrence of a gaping anus as a significant symptom.

The disease begins with colicky pain and this may be repeated in mild form from time to time. It is in the nature of cramp-like spasms of short duration. It is usually possible to trace back the beginning of the intussusception to an initial vomiting, which may persist for a few days, and reappear at times. The temperature remains normal. The diagnosis is made upon the finding of the cylindrical (sausage-shaped) tumor, usually lying transversely in the upper abdomen just below the liver. This tumor may be mistaken for the thickened omental mass found in the same region during the course of tuberculous peritonitis. In both chronic intussusception and tuberculous peritonitis there is progressive loss of weight. Still thinks it possible to detect a variation in the consistency of the palpated mass which at one moment may be hard and at another soft. Dance's sign may be present, though it would seem to require considerable experience to recognize it. It is characterized by a lack of fullness (unnatural emptiness) in the right iliac fossa.

The pathological changes are less pronounced than in the acute type. The slow course allows the stretching of the peritoneal attachments and



mesentery, and in this way the circulation in the invaginated bowel is only slightly affected. There is some edema and swelling of the intestines and in some cases fibrous and dense adhesions develop between the invaginating surfaces or surrounding mesentery. Hypertrophy and dilatation of the intestinal wall proximal to the intussusception have been described.

*Recurrence.*—In occasional instances the intussusception recurs and requires re-operation. Four such cases were found in forty-one reported by Cohen. He emphasizes the importance of detecting recurrence in any case that has previously been operated upon, because the operation is only palliative and not radical.

*Diagnosis.*—*Differentiation.*—Probably the disease is most often confused with ileocolitis. Exclusively breast-fed babies seldom have an enteritis and certainly never with bloody discharge as one of the first symptoms. The stools are watery and contain much mucus, and while blood is a common manifestation, it is not so acute in appearance.

Henoch's purpura is accompanied by rash of a petechial nature, by joint pain, by albumin and blood in the urine. It is found in children past the age susceptible to acute intussusception. Abdominal pain and fresh hemorrhage are present and in this regard the two diseases are similar.

Hemorrhage from intestinal ulceration, from pin-worms or from rectal prolapse is accompanied by none of the other characteristic symptoms of intussusception.

In the severe cases of bronchopneumonia in infancy, marked tympanites with meteorism and constipation appearing suddenly may give the suggestion of obstruction from invagination.

The absence of one or more of the typical symptoms does not exclude the presence of an invagination. Exceptionally a tumor may be felt per rectum before the appearance of hemorrhage. In any event rectal exploration is indicated at all stages in cases having a history of acute colic.

*Treatment.*—This is obviously entirely surgical. It should be instituted at the earliest moment following the diagnosis. It is important that the physician to whom the case is first brought should recognize the surgical aspect of the case. In all instances of abdominal pain or hemorrhagic stools a tumor should be suspected and its presence searched for during a careful physical examination. As one looks back on such cases he resolves to get the assistance of the surgeon earlier the next time.

The character of the surgical treatment is influenced by the age of the pathological lesions and their extent. Edema and swollen mesenteric glands are common; adhesions are rare. The inclusion of the mesentery with swollen lymph-nodes makes the reduction of the invaginated bowel difficult and in some cases impossible. Strangulation which is irreducible and gangrene due to obstruction of the circulation call for a resection of the affected portion. This is a grave matter in childhood, usually resulting fatally. Thompson reported six deaths in ten cases in whom resection was done.

The surgery of the subacute and chronic types offers much better outlook. Resection has not been found necessary and nearly all cases have recovered.

**Prognosis.**—By the time that edema, mesenteric adenitis and necrosis have developed the reduction of the invagination is difficult. Simple early reduction is successful, while resection usually results fatally. The mortality increases with the duration of the disease, but even at the end of twenty-four hours is considerable. Eugene H. Smith found that the infants who recovered had been operated upon in from eight to twenty hours after the onset of symptoms. Brown reported thirty-one cases, one-third of whom died following operation within the first twenty-four hours. The figures and conclusions of Ladd are convincing. He found that in recent years the mortality following early operation has become much lowered. In various Boston hospitals the study of the statistics of sixty-three cases of the acute type showed no death when operation had been done within the first twelve hours, but the mortality rapidly increases thereafter, 16.6 per cent in the second twelve hours, 23 per cent in the third, 54 per cent in the fourth and 88 per cent after sixty hours.

## PERITONITIS

**Fetal.**—In fetal life a rare chronic form of peritonitis may occur. It is sometimes ascribed to the absorption of toxins and infections from the mother. Syphilis undoubtedly may cause it. Malformations of the intestines such as stenosis, atresia, volvulus and kinking, may be connected with fetal peritonitis. Westphal reports such a case in a premature infant dying on the second day. The abdomen in his case was markedly distended and the skin shiny. An examination showed no infection, but a peritonitis, caused by congenital atresia of the duodenum and a fusing of the jejunum and ileum.

When peritonitis exists in the infant at birth, the symptoms are much the same as found in the later appearance of the disease. The abdomen is greatly distended, there is tympany of the intestines and fluid in the peritoneal cavity. The increased intra-abdominal pressure causes difficult breathing and cyanosis. Treatment is of no avail, and the infant does not long survive birth.

**In Early Infancy.**—The newly born infant is peculiarly susceptible to infections and does not bear them well. Sepsis is particularly liable to occur therefrom. Infection and suppuration of the navel may result in the inflammation of the umbilical vessels, and transmission thereby. Erysipelas of the genitals or of the abdominal wall may cause peritonitis. Intestinal rupture, resulting spontaneously or from birth trauma, has been reported and it is followed by peritonitis. Intestinal ulceration and perforation in the first few days of life have been reported, and the resulting

contamination of the peritoneum by meconium or feces causes death in a few days.

Peritonitis in the newly born is recognized by the abdominal tenderness; distention, rigidity, protrusion of the navel, drawing up of the legs, and by prostration. The temperature is always above the normal, and usually markedly elevated. The blood count, when high and mostly polymorphonuclears, is of diagnostic value. The outcome is always fatal.

The abdominal viscera are covered with fibrinous adhesions and pus, and the abdominal distention is due to the presence of gas in the intestine and to accumulation of peritoneal fluid.

**In Later Infancy and Childhood.**—The most common cause is tuberculosis, which furnishes the chronic type found in childhood. It will be discussed in Chapter XXII.

Acute peritonitis is usually due to acute intestinal lesions, especially intussusception, appendicitis, wound or other traumata. Very rarely the ulcers of dysentery and typhoid may undergo perforation with spread of infection. Cases in which no origin outside of the peritoneum can be found are regarded as idiopathic and primary, though it is probable that the focus is overlooked, and that the infection is carried through the circulation. The infective organism is commonly the pneumococcus or streptococcus, occasionally a staphylococcus or colon bacillus. It is especially difficult to trace the source of the pneumococcic infection. It is probable that it is carried by the blood stream or that it is absorbed from swallowed sputum in the intestine. Streptococci are carried in these same ways from the tonsil or ear. The statement of McCartney, that primary pneumococcic peritonitis occurs only in females, is of interest. I have seen one case, however, in a boy.

Rare cases are found mentioned from time to time in the literature. The disease is occasionally due to such infections as scarlet fever. The hemolytic streptococcus has been recovered from the peritoneal exudate in such instances.

Peritonitis in childhood is usually diffuse. Localized peritonitis or abscess is of rare occurrence. It may develop in older children as a walled-off process around the appendix, rectum or above the liver. Subdiaphragmatic abscess may be due to direct extension of pneumonia or a purulent pleurisy. Vaginal or gonorrheal infection may cause a pelvic peritonitis. In intussusception there may be an inflammation of the peritoneum limited to the affected intestine; it may result in general involvement of the peritoneal cavity; in numerous instances no peritonitis may be present.

**Latent Peritonitis.**—It is not infrequent at autopsy to find an unexpected purulent exudate in the peritoneal cavity of emaciated infants. Foote has designated this as "static" peritonitis. It may be found as a terminal complication of marasmus, lues, tuberculosis and other states where the nutrition and resistance are lowered. In these malnourished



infants all of the vital functions are depressed. There is a decreased amount of blood and corpuscles in the circulation as a result of which the tissues suffer by starvation. As a result the peritoneum cannot avoid infection, and by the feeble reaction to such invasions gives no active manifestation of its presence. This occurs not infrequently in undernourished infants who have undergone some intestinal operation such as pyloric stenosis. Such an outcome is one of the reasons for the increased operative risk in emaciated subjects.

The local symptoms are few or so insignificant as to be overlooked. The child does not show any evidence of pain by crying, but it is suggested by a drawing-up of the legs. Distention is present but is not so noticeable as found in the peritonitis of robust children, nor is there evidence of intestinal paralysis. The temperature shows no marked reaction by elevation. The white blood count is not greatly increased, but the polymorphonuclear white cells are proportionately more numerous. The preëxistence of a long-continued marasmus or loss of weight should cause one to place a proper emphasis upon the appearance of even slight abdominal distention, tenderness and the other obscure manifestations which these infants may present, but these indefinite symptoms may be unrecognizable. At laparotomy a fibrinous or purulent exudate will be found, but the primary focus not discoverable. The streptococcus or other pus-producing organisms are present. Practically all cases are discovered only at postmortem.

**Streptococcic Variety.**—This type in children is of striking appearance, marked by acute severe pain, local tenderness, general rigidity and distention of the abdomen, continuously high fever and in some cases delirium. There are usually vomiting and diarrhea. The picture is one of sepsis. The urine is scanty, contains albumin and is of high specific gravity. The white blood count, especially the polymorphonuclears, becomes progressively higher. The outcome is fatal because of the overwhelming nature of the infection.

The disease is more apt to occur during the season of epidemic diseases. There is usually history of a recent or existing sore-throat, though the infection may come from a throat in which there has been no recognized inflammation. In either event, the route of transmission is hematogenous. The severe type of angina, seen with or without a scarlet-fever rash, and due to the streptococcus, is the most apt to cause the disease, though any streptococcic focus may likewise introduce the organisms into the circulation. When the case comes to operation, pus is found in the peritoneal cavity.

At autopsy the abdominal viscera show participation in the toxic degeneration, and the intestines are matted together by adhesions and a plastic exudate. The infection is widespread, and the lungs, pleura, bronchi and pericardium show inflammatory and purulent changes.

**Treatment.**—Because of the persistent vomiting, the stomach should be washed out daily with plain cold water, some of which should not be with-



drawn. Saline solution should be given also by hypodermoclysis in the region below the breasts.

In a blood-borne disease of this seriousness, the early use of intravenous therapy is indicated. It is too soon to decide as to the safety and efficacy of the newer remedies, gentian-violet or mercurochrome. Antistreptococcus serum has been disappointing, but it is probably due to the fact that in this rapidly fatal disease infection is spread to numerous other viscera by the time serum is injected. No harm can come from a suitable blood transfusion which should be performed as early as possible in the disease. It warrants a trial.

The case is referred to the surgeon because of its resemblance to the peritonitis of a ruptured appendix. Abdominal drainage is all that can be done in the way of operative relief. This does not effect a cure, possibly because of the delay in the procedure.

**Pneumococcic Variety.**—In cases dependent upon a preëxisting pneumonia or empyema, the development of peritonitis may be masked by the preëxistence of the fever and respiratory symptoms and by the gradual onset. Other cases have acute and stormy symptoms beginning with high fever, severe vomiting and evidences of abdominal involvement. The stools are not normal—in some cases diarrheal, in others constipated. The palpation of the abdomen reveals definite local or general tenderness, but there is little evidence of muscular rigidity. The presence of fluid may not at first be suspected, but gradually the abdomen becomes more enlarged and distended and a diffuse fluid wave is discovered. In other cases a definite mass may be found in the region of the umbilicus or hypogastrium and this may be recognized later by rectal examination. There is always a leukocytosis with an increase above 60 per cent in the polymorphonuclears. As a rule the course is not so rapid as a streptococcic infection, but is more so than the tuberculous variety. Coexisting pulmonary disease may be found and the other serous cavities of the body may be involved, discovered at autopsy.

**Etiology.**—The incidence of the pneumococcus as a cause of peritonitis of children is about 5 per cent. Beaven found that nine children in a total of 171 cases of peritonitis were infected with the pneumococcus. Infection seems to accompany respiratory diseases or to follow soon afterwards, in some cases before the cough has disappeared. Pneumococcic otitis media may broadcast the organisms so that they find lodgment in the peritoneum. Mention has already been made of the occurrence of infection by way of the intestines, a mode which is difficult to prove in the absence of lesions in the intestinal wall, but entirely possible by the absorption of viable micro-organisms which have reached the intestine in the sputum.

**Diagnosis.**—Abdominal puncture is performed sometimes for determining the presence and character of the peritoneal fluid. This procedure is not necessary when the presence of fluid is obvious, and it is better to

promptly drain the peritoneum. The difficulty of differentiating the abdominal distention of pneumonia from pneumonia with coexisting peritonitis is obvious, but fortunately rarely encountered. The occurrence of acute vomiting, of abdominal pain and the appearance of tenderness in the abdomen furnish symptoms which are not usually present in pneumonia.

The diagnosis from appendicitis is also difficult. Early in appendicitis, it may be possible by examination of the child to find the characteristic pain and tumor in the right lower quadrant, following which a diffuse peritonitis usually develops. Pneumococcic infection of the peritoneum is usually diffuse from the beginning.

An unsuspected case of pneumococcic peritonitis occurred in a seven-year-old boy with chronic nephritis and general edema. His history is as follows:

After an attack of scarlet fever the boy developed intermittent general edema, but was free from it for a period of one year. He was brought into the hospital twenty months after the onset, with a return of symptoms. His face and extremities were swollen, the abdomen ascitic, the urine almost suppressed and full of albumin and granular casts, the blood-pressure 120 over 80. The scrotum was edematous and the abdomen distended and slightly tender. The output of urine was one-fifth the intake. The stools were kept open with salines. The phthalein secretion was 15 per cent at two hours, and 45 per cent at four. Cyanosis and air hunger were pronounced just before death. At autopsy the peritoneal fluid was found to be milky, purulent, due to infection by the pneumococcus. The pathologist regarded the peritonitis as primary, with both acute and subacute lesions. The kidneys showed both acute and chronic diffuse nephritis.

The milky fluid obtained from the peritoneum resembles that of chyle. Chylous ascites consists of peritoneal fluid which contains much fat. This condition is rare in childhood.

*Prognosis.*—In pneumococcic peritonitis alone the prognosis is favorable. When there has been a primary pneumonia or when the infection has spread to the pleura and pericardium, the outcome is usually fatal.

*Treatment.*—As soon as it is discovered, the purulent fluid is to be drained, whether it is general or circumscribed as a localized abscess. The administration of water in any way that can be retained is necessary. Abdominal pain may require an occasional dose of paregoric.

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## CHAPTER XXI

### ABDOMINAL EXAMINATION AND CERTAIN DISTURBANCES OF THE ABDOMEN

**Abdominal Examination.**—With the child who is old enough to become frightened or to resist the attempt at examination, it is well to first get acquainted. Most little folk will soon be friendly if the physician is not too brusque or too serious in his demeanor. Those with an unstable nervous make-up will probably have a tantrum in spite of any method of approach, and I have seen a few who never became reconciled. Getting the child's confidence and consent is a necessary preliminary to stripping him of his clothing for the examination of the abdomen. It is well to direct his attention to something foreign while the abdomen is being palpated, or to employ some device such as suggesting that one can feel what he has eaten for breakfast.

The child that screams and clings to the mother's arms cannot be examined until it can be induced to lie quietly on the bed or table. It is better in such cases to have some one besides the parents assist in managing the patient. Fortunately for one's success in the undertaking, the examination of sick or prostrated children meets with few difficulties.

If the child is able to stand, it is helpful to observe him in this position, for it discloses the presence of inguinal hernia, a prominent or sagging abdomen, and the shifting nature of fluid contents. While the patient is leaning forward the abdominal muscles are relaxed and palpation made easier.

The objective findings are not difficult to obtain when the abdominal wall is soft, which is the natural tendency in children. Differentiation is simple in childhood, if one be familiar with the diseases peculiar to this time of life. Malignancies and diseases of the genito-urinary tract which are common in the adult abdomen seldom come into question.

*Inspection.*—The child should be placed in a good light. The size and level of the abdomen should be noted. Abdominal prominence in thin children is always of marked significance, due to numerous causes: inflammation, dilatation of the hollow viscera, visceroptosis, ascites or tumors. Gaseous distention is most common in intestinal indigestion and obstruction, but it is present in peritonitis and often in pneumonia. The chronically enlarged abdomen may be due to rickets, Hirschsprung's disease of the colon or celiac disease. The abdomen steadily enlarges in abdominal



tuberculosis. Acute distention causes grunting and embarrassment of the respiration.

Fullness of one portion of the abdomen or emptiness of another should be noted, a phenomenon especially of diaphragmatic hernia, dilated stomach and intestinal obstruction. Umbilical and inguinal hernia are easily seen, the disappearance of the latter during the recumbent posture being of diagnostic importance.

Visible peristalsis and intestinal patterns due to spasm of the muscular coats or to obstruction are usually well outlined. Normal peristalsis is commonly seen through the atonic wall of emaciated infants. Engorgement of the veins and obliteration of the umbilicus occur in ascites.

Scaphoid or retracted abdominal wall is the rule in marasmus, cases of tuberculous meningitis unless complicated by peritonitis, and in the intestinal infections characterized by marked dehydration.

The attitude of the child is characteristic when abdominal pain is present. The young infant screams and draws up the knees in the attempt to relieve the pain from muscular spasm and distention. Older children lie quietly and the legs are kept flexed.

Hemorrhages of the skin, especially petechiæ, will be found in Henoch's purpura, suggesting the nature of the associated intestinal bleeding.

Small necrotic papules of the skin may be seen in advanced cases of general miliary tuberculosis.

*Palpation.*—Palpation should be begun slowly and not until the child has gotten somewhat accustomed to the physician's presence. The hands of the examiner should be clean, warm and applied gently. The abdominal contents of children are relatively easily felt unless there be a physical barrier such as rigidity, muscular spasm, gaseous distention or inflammation. Much can be learned about the tension of the abdominal wall, areas of pain, mesenteric nodules, obstructive tumors, pyloric hypertrophy, fecal masses, enlargement of the liver, spleen and kidneys. Importance should be placed upon the finding of areas of tenderness, especially with evidence of flinching, which is a more reliable evidence of tenderness than questioning the child. The examiner must learn to make allowances for unreliability in subjective symptoms, especially the child's answers to queries about painful areas.

Involuntary contractions of the abdominal wall may be caused by tickling or forcible handling while the abdomen is being palpated. Seriously sick children seldom show the tickling reflex. Local spasm of the abdominal wall resembling a tumor mass is sometimes encountered during the examination, due to reflexes passing from the viscera to the abdominal muscles.

Fluids are usually due to peritonitis, most commonly of tuberculous origin. In some cases ascites in children is due to nephritis, but is rarely caused by cardiac or hepatic disease. Abscesses complicate appendicitis, perinephritis, vertebral and mesenteric tuberculosis.

Palpation is of great service in the early diagnosis of appendicitis when

local spasm of the overlying muscles is felt and much tenderness is evidenced. Later the general soreness and rigidity over the abdomen discloses the diffuse peritonitis which commonly develops.

The edge of the liver is normally felt slightly below the costal margin during infancy. Seldom is it sufficiently reduced in size to escape palpation on deep inspiration. Fatty liver is common in the newly born and in chronic infections of childhood, and the hard edge may be felt an inch or more below the edge of the ribs.

The spleen should be examined in every abdominal palpation, with the physician standing on the right side. The anterior ends of the lower ribs are easily mistaken for the spleen if one does not isolate them carefully. When the spleen is enlarged it is equally so in a downward direction, and freely movable both on deep inspiration and by palpation. The common causes of enlargement are toxemia, syphilis, typhoid fever, malaria, anemias and tuberculosis.

Tuberculous mesenteric glands may be felt anywhere in the abdomen, but particularly in the right lower quadrant, under the liver or in the neighborhood of the umbilicus.

*Percussion.*—Fluid in the abdomen is determined by combined palpation and percussion. The fluid wave is obtained by deep pressure of the edge of the hand in the median line of the abdomen and tapping the flank with the fingers of the opposite hand. The presence of fluid is manifested by a rebound against the examining hand.

The liver in children, though best found by feeling the edge of the lower border, may, in a distended abdomen, be disclosed only by percussion. Liver dullness disappears in inflammatory lesions which are accompanied by exudates and peritonitis.

A distended urinary bladder is sometimes found in children, easily detected by palpation or percussion.

**Causes of Obscure Abdominal Pain.**—Acute abdominal pain should be promptly considered as surgical in nature until proven otherwise. The benign, obscure or rare types of pain are not so urgent, but require thorough study in the attempt to find the cause and treatment.

Causes outside of the intestinal tract may be difficult to locate. In the first place the inability of young children to determine the location of the pain, and their tendency to the unreliable because of suggestion, make the diagnosis a difficult matter.

One should bear in mind the clinical observation that abdominal discomfort may occur from infections in distant parts. Infections of the tonsils, ears, cervical lymph glands, febrile respiratory diseases, especially influenza, will sometimes be accompanied by puzzling attacks of pain ascribed to the abdominal region. It is probable that systemic infections, possibly originating in the throat, may, as suggested by Brennemann, produce enlargement and inflammation of the mesenteric glands and abdominal

pain. Involvement of the lymph-nodes in this manner is to be differentiated from suppurative or tuberculous disease. Infections outside of the intestinal tract do cause obvious indigestion, abnormal stools and discomfort. That pain may be referred in a reflex manner to the abdomen should be considered only after all other causes are excluded.

Recurring attacks of colic in the region of the umbilicus are sometimes found in children. The history is suggestive of recurrent appendicitis, but at operation lesions of the appendix are not found. I know of no way to find the origin of these not infrequent complaints of children after the usual methods of exclusion have been employed. Parasites which migrate to the region of the biliary tract are probably more frequently the cause of such pain than we can prove. There is increasing evidence that low types of parasites, such as the *Balantidium coli* and the *Lambia*, do produce intestinal symptoms, which may be indefinite but usually consist of discomfort or occasional pain of the colicky type. As mentioned elsewhere, allergy may be manifested by abdominal pain. Pyelitis, common in girls, may have tenderness and pain in the region of the kidney.

Biliary disease as a cause is so rare in childhood that it is seldom suspected, and only found at operation or autopsy. Still has found ten instances of minute stones at autopsy in infants, death occurring during the first month of life, and the lithiasis probably congenital. In older children gall-stones are seldom present. Cholecystitis is reported very rarely, the disease causing vomiting and epigastric pain. Distention of the gall-bladder due to infection with typhoid or paratyphoid bacilli, with or without the presence of stones, has been found in children. Pain is the most constant and often the only symptom.

**Visceral Ptosis.**—This is one of the most frequent causes for chronic gastro-intestinal disturbances. It is most common after the age of three years and is to be expected in the child of thin, lanky type with pendulous abdomen, flat chest, stooped shoulders, lordosis, semiflexed knees and flat feet. The child tires easily and his general appearance indicates a fatigue posture and poor nutrition. The rounded shoulders and exaggerated spinal curves are compensatory.

Children of this type have a relaxed abdominal wall, and the gastro-intestinal musculature is atonic, permitting sagging of the stomach, intestines and other abdominal viscera. The circumference of the abdomen below the umbilicus is much larger than the upper abdomen. Poor bodily mechanics interfere with the digestive function and thereby reduce the nutrition and body weight.

Another type of child is less commonly observed, in whom the abdomen is short, with the position of the diaphragm, stomach and bowels lower than normal, but with the same flabby muscles and the functional derangements of the organs.

The influence of poor posture upon the health of children has become



more generally recognized in recent years, the interest in the subject having been greatly stimulated by the reports of large numbers of physical defectives among the boys enlisting for the army. This type of child is familiar to the orthopedist and pediatrician, and deserves a larger recognition and attention by all physicians who deal with youth.

*Manifestations.*—A vicious circle is present in the typical case. By the injurious effects of poor ventilation of the nose and throat, chronic infection of the upper respiratory tract, glandular hyperplasia and many other disturbances, the appetite suffers and the nutrition becomes impaired. The child tires easily and does not hold his body erect as he does in health. In many cases there is a familial tendency to the “lanky” type of build which is a predisposing factor. Such individuals do not well digest or utilize fats. The end result is relaxation and displacement of the stomach and intestines, as a result of which there is further impairment of appetite, failure of the stomach to empty completely or on time, vomiting and constipation. The stasis often produces a low-grade toxemia which has its effect upon the general health.

There are three disturbances resulting from visceroptosis which need emphasizing. The most common is constipation. That ptosis has much to do with this is shown by the immediate improvement following the wearing of proper abdominal supports and the correction of the body posture. The sagging, kinking and the redundancy which is sometimes present in the large intestine interfere with the



FIG. 46.—FIVE-YEAR-OLD GIRL WITH VISCEROPTOTIC ABDOMEN AND SLUMPING POSTURE

Note the back knees and the stooped shoulders. This child had constipation, attacks of recurrent vomiting, underweight and underheight.



mechanism of emptying. The colon is shown by the roentgen examination to be elongated, tortuous and dilated, especially in the sigmoid region.

A less frequent manifestation is that of abdominal discomfort and sub-acute pain. Retained feces and the weight of the organs produce dragging and stretching of the supports. The pain is sometimes of a colicky nature, probably caused by dilatation of the intestine.

Vomiting may be caused by pyloric irritability or stasis, as a result of the marked angulation of the pylorus and duodenum. Undoubtedly the recurrent type of vomiting may be due to the mechanical disturbance produced by visceroptosis. This is indicated by the usual disappearance of symptoms when the stomach and intestines are supported in their proper position.

*Treatment.*—A great advance has been made in the restoration of the well-being of children by the recent methods not only for correcting the body posture, but thereby bringing the child's digestive tract up to the normal.

As the child becomes tired easily, he requires rest, and in the beginning this should be obtained by putting him to bed for a week or more. Cases with marked fatigue posture should have particular attention paid to the details. Brown and Talbot have suggested a method of treatment which I have found to be practicable in the home. While the child is in bed a pillow is kept under the lower dorsal and lumbar vertebra. Simple breathing exercises are necessary for expanding the chest. These are performed with the child in the correct posture. The child is taught to stand correctly by contracting the gluteal and abdominal muscles, which will take the strain from the mesentery by upward pressure upon the intestines. Several times daily he should practice standing with the head held up, the chin depressed, and all of the posterior surface of the body touching a straight surface such as the wall. When children do not respond to such educational methods of correction, additional support should be obtained by a combined shoulder brace and abdominal corset. I have found that the mother or some good seamstress can be taught to make a comfortable and effective corset (see constipation), which if worn during the active part of the day promptly assists in correcting constipation, posture, appetite and nutrition. Recurrent vomiting is strikingly relieved by permanent postural correction. The bowel should be unloaded by regular administration of mineral oil until the function is corrected. Proper diet aids in the cure of constipation and the improvement of intestinal tone. The appetite and nutrition begin to improve at once, these children putting on remarkable amounts of weight. The change from a puny ailing child is a matter of pride to many of these youngsters, and I have found that they coöperate in all the curative régime in spite of the fact that apparatus must be worn. From three to six months are advisable for active treatment before correct habits are firmly established.

**Abdominal Purpura.**—One of the definite though rare manifestations of intestinal hemorrhage is Henoch's purpura, a syndrome belonging to purpura hæmorrhagica. Search for other evidence of bleeding will result in a positive history of such a tendency, or in the actual discovery of petechia, ecchymoses and other hemorrhages.

The general disease, purpura hæmorrhagica, was first described by Werlhoff who gave it the name of morbus maculosus. The abdominal type was designated as purpura abdominalis by Henoch in 1874.

Purpura is seldom seen in infancy. Most cases come under observation when from three to five years old. However, as the disease is of extended duration, all ages are affected. It appears from statistics to be more common in girls.

Essential purpura, characterized by its intermittent nature, is a definite disease due to a blood deficiency, known as thrombopenia. The thromboplastic or fibrin-forming material of the tissues is poor, and the production of platelets by the bone marrow is deficient. The cause which underlies this deficiency is not known but it is probably congenital.

Infectious purpura has an entirely different etiology. Cases of this nature are grouped under the name of symptomatic purpura, and follow an infection, usually a septic sore-throat or scarlet fever. It may also be due to diphtheria or tuberculosis, and I have seen three cases of intestinal hemorrhage, persistent for several days, following measles.

*Symptoms.*—The abdominal symptoms are somewhat similar to appendicitis and intussusception. Abdominal pain of a colic-like nature, associated with vomiting and bloody stools, certainly suggests an invagination of the bowel, especially when there is board-like rigidity and tenderness of the abdomen. The tenderness is usually found in the umbilical region, or in the location of the transverse colon. An abdominal mass may be palpable. The vomiting of blood or the occurrence of hemorrhagic stools should make one inquire about previous similar attacks and the history of purpuric lesions. There may have been swollen, hemorrhagic joints.

The Henoch syndrome follows suddenly after the eruption of the skin lesions. Petechia and ecchymoses occur on the extremities and the buttocks. There may be bleeding from the nose or gums, and blood in the urine. The intestinal hemorrhage comes chiefly from the colon. In addition to this free blood, there is spontaneous bleeding into the coats of the intestines and peritoneum, and at times into the pleura and lungs.

The essential nature is one of thrombopenia: a marked reduction in the number of blood platelets, with a prolonged bleeding but not disturbed coagulation time. However, the clot does not retract promptly. The course of the untreated cases of essential intermittent purpura is to become worse. Fever and convulsions may be present in young children during the abdominal crises. The hemorrhages last for a week or more, to disappear and return at indefinite periods.

*Diagnosis.*—The mistake is commonly made of regarding abdominal purpura as a surgical disease. The acute attack of vomiting and colic at once suggests appendicitis, while the sudden appearance of pain, vomiting and bloody stools resembles intussusception. Operation has been performed without the general hemorrhagic nature of the disease being suspected, and to one's surprise, surgical disturbance not found.

The following case is an illustration of such an accident:

A seven-year-old boy had been having rheumatism and joint symptoms for three weeks, not relieved by salicylates but intermittent in character. Suddenly he began to pass blood by the stools, associated with much pain, these symptoms persisting for three days, for which reason he was brought into the hospital. A diagnosis of ulcerative colitis had been made, but as the manifestations seemed to point towards appendicitis, operation was performed. A normal appendix was found and no evidence of intestinal disturbance. Within a few days, numerous hemorrhagic lesions were discovered upon various portions of the body. Not until this time had the purpuric nature of the disease been suspected.

Laparotomy is unnecessary and of course contra-indicated. A careful history, a general examination of the child and the determination of the bleeding time and the number of the blood-platelets will be of great assistance in making the diagnosis.

Because of the difference in the prognosis it is important to determine whether hemorrhage is due to purpura or to hemophilia. A summary of the differential points shows in:

<i>Purpura hæmorrhagica</i>	<i>Hemophilia</i>
Absence of hemophilic family history	Definite history of disease transmitted through females
Spontaneous hemorrhage from skin, mucous membrane, kidneys and intestine	No purpura or mucous membrane hemorrhage. Bleeding or ecchymosis occurs from trauma
Coagulation time normal or only slightly lengthened	Coagulation time prolonged
Bleeding time prolonged	Normal bleeding time
Clot soft and not well retracted	Clot finally forms and may not be completely retracted
Absence or reduction in blood-platelets, and when below 30,000 visceral hemorrhages may occur	Platelets normal or increased

The treatment is by repeated blood transfusions from a suitable donor every three to five days during recurrences. From our experience in the Kansas University Hospital, I am convinced that this is beneficial. In essential purpura we have begun the practice of removal of the spleen, which is now recognized as necessary for the cure. Operation should be done, following a course of transfusions, at a time when the symptoms are absent.

The essential purpuræ are not benefited by any serum or hemostatic agents which I have seen tried. When these remedies are successful, the disease is not due to thrombopenia.

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## CHAPTER XXII

### ABDOMINAL TUBERCULOSIS

Invasion of the abdomen is common among the children affected with tuberculosis, and the infection involves three important structures; namely, the intestine, the peritoneum and the mesenteric lymph-nodes. It is more practical to designate the disease as abdominal tuberculosis, for seldom except in the beginning is the process localized to one alone of these structures. The pathological lesions pass through progressive stages, all of a tuberculous nature, with, however, secondary pyogenic infection when perforation of the intestinal wall has occurred.

The lesions may be more prominent in one structure, causing manifestations which will be directed to that region. For this reason it has become the custom to speak of tuberculosis of the intestine when the leading symptom is ulceration and hemorrhage, of mesenteric tuberculosis when the lymph-nodes of the mesentery are hyperplastic or caseous, and peritoneal tuberculosis when the abdomen is greatly enlarged, due to the accumulation of ascitic fluid or the matting together of the intestines, omentum and mesentery by a fibrous or adhesive exudate.

In practice among children there are three chief difficulties which we encounter in dealing with this disease. The usual insidious course of development delays the recognition both of the abdominal nature of the affection and the fact that it is tuberculous in character. Further, one cannot by clinical observation alone know in which structure the abdominal invasion first occurs. In fact, as stated above, we should forget the usual classification and regard all the organs mentioned as involved to a greater or less extent, or as secondary to tuberculosis within the thorax. Lastly, special local treatment should be left out of consideration until the general measures which are applicable to all stages and manifestations of tuberculosis have been followed. The chief indications for surgical intervention are acute intestinal obstruction and perforation with abscess or secondary peritonitis. In general we can conclude that these children need only medical treatment unless complications of a surgical nature arise.

Tuberculosis of the abdomen in children is always to be regarded as serious because of the fatal outcome from local catastrophies, or from the dissemination or appearance of the infection in other portions of the body. Early recognition and proper treatment is necessary for a cure, or for limiting the spread of the lesions. Much can be done in mitigating pain and in increasing the comfort and nutrition of the child.

**Occurrence in Childhood.**—Invasion of the abdominal viscera is most common at the age in which pulmonary and glandular tuberculosis is most prevalent. This is usually found to be between the first and fifth years, when resistance is lowest. Gradually as the child grows older this resistance increases and the disease is not so frequent. Exciting factors are found in infected milk and in the respiratory contagious diseases which are most often encountered at this time of life.

That the disease is far more common than is clinically suspected is shown by the discovery at autopsy of tuberculous lesions when the child has died of either unrecognized tuberculosis or of some other immediate cause. As an illustration of this might be mentioned the relative frequency with which tuberculous lesions are found at autopsy by numerous pathologists in children dying of diphtheria in as high as 20 per cent of these deaths. In about one-third of these instances of tuberculous infection, the abdominal tract will be involved. Intercurrent disease therefore may terminate life before such tuberculous infection has time to produce symptoms which might be recognized.

It is only in hospital admissions that we can learn much about the incidence of abdominal tuberculosis. Here the frequency will depend upon the hygienic conditions of the surrounding country, and the extent to which the general population is affected. The widespread use of unboiled and infected milk permits of a high incidence. This is shown in the death-rate from tuberculous peritonitis in New York City, where eight of each one hundred thousand children formerly died of this disease, but now, with sterilization of the milk supply and with exclusion by physical examination of infected food handlers, there is only one death from this cause in the same number of children. In communities where adults suffering from tuberculosis are commonly at large, the children are easily infected and the occurrence is high. In the general run of hospital children, I have noted the report of abdominal tuberculosis as one to each 500 admissions, while in Australia it is as rare as one in each 6,500 admissions.

Tuberculous peritonitis is found in 10 per cent of tuberculous children under two years of age, but it is more frequent than this in somewhat older children, up to four years of age. It occurs in infancy not infrequently.

Intestinal tuberculosis without peritonitis occurs once in every 280 tuberculous children.

Ascites is demonstrable in one-third of the cases of tuberculous peritonitis, adhesions in nearly one-half, and ulcerative lesions of the intestines in the remainder.

Mesenteric tuberculosis is more frequently discovered than the other types of lesions.

**Etiology and Pathogenesis.**—Primary tuberculosis, or the disease confined chiefly to the intestinal tract, is found more frequently with children. While the disease may exist without known pulmonary lesions, it is probable

that in nearly all cases a focus exists outside of the abdomen. Certainly in generalized miliary tuberculosis, the infection is present to some degree in the abdomen.

The causative organism is more frequently of bovine origin, in which case it reaches the intestine most usually in the milk. The human type of the tubercle bacillus gains entrance in the sputum swallowed after coughing, or by way of the blood stream and lymph channels from the tonsils, cervical glands, bronchial glands or lungs.

The specific nature of the infection in intestinal tuberculosis is proven by the repeated isolation of tubercle bacilli from the stools. There has been much difference of opinion as to the occurrence of primary infection of the intestinal mucosa, but for practical purposes it must be regarded as possible. In the young infant, such as I have known, who has never been out of the house, and never exposed to human tuberculosis, but has been fed unboiled cow's milk, and has had no previous illness, the origin of tuberculous peritonitis must occur in some way through the intestinal wall, whether by a mucous membrane lesion or by direct absorption through the lymphatic follicles.

Primary infection takes place from raw milk or from meat. Any milk even from tested animals may become infected. In countries where the milk is boiled as a routine, abdominal tuberculosis is not so common. Rare cases of infection have been ascribed to the swallowing of bacilli from diseased nipples of the mother or wet-nurse. However, in every case of tuberculosis in a child, the parents or other members of the household should be suspected as spreading the infection until proven negative. Following direct exposure the child develops infection by the air passages, and abdominal tuberculosis becomes secondary to the disease in the lung and bronchial glands, the bacilli being carried by the blood or lymph stream. The micro-organisms may be deposited in the lymph follicles of the intestine, which is suggested by the occurrence of deep ulcers of these structures. Some writers deny that ulcers can be produced by the action of bacilli introduced through the food or swallowed sputum, but there can be no difference of opinion as to the existence of such lesions, for they are present in generalized tuberculosis, and are common in cases dying therefrom. Ghon, in a series of autopsies in children, found no proof of infection beginning in the intestine, but involvement of the mesenteric glands was found at autopsy as frequently as that of the bronchial glands; such findings leave the question unsettled.

In general one concludes that the infection is spread from the tonsil, the bronchial glands or the lungs; that in the absence of disease in these structures and when infected food has been used the disease has originated from intestinal absorption. About 50 per cent of abdominal tuberculosis is regarded as due to infection from the bovine type of bacillus by way of the food. However, all individuals are exposed to inhalation of bacilli, whether

bovine or human, from air and dust, and to swallowing of the same in contaminated food.

The influence of season has much to do with the development of the disease. From January to March respiratory epidemics are common and these are soon followed, usually up till June, by the more frequent occurrence of tuberculosis.

It is possible that bacilli may reach the peritoneum through the intestinal wall without a tuberculous lesion being present, but because of injury from some intestinal disease the wall becomes pervious to the microörganism. This is undoubtedly true when infected milk is fed to the child with acute intestinal indigestion or convalescent therefrom. Peritonitis as the primary tuberculous lesion in the body probably never occurs. It is more probable that the disease reaches the peritoneum from the circulation, or by direct extension from disease in the intestine or lymph gland. Latent disease in the lungs or glands may be lighted up and spread following measles, whooping-cough or influenza. The lungs are more frequently associated with tuberculous peritonitis than is the intestine, but the tendency of children to swallow sputum offers an explanation of the more common occurrence of abdominal tuberculosis in young individuals. The route is most commonly through the lymphatic circulation.

*Intestinal Lesions.*—Multiple ulceration is the characteristic lesion. Ulcers may occur anywhere in the intestinal tract, but the favorable site is the ileum close to and sometimes involving the cecum, at first appearing in the lymph follicles (Peyer's patches). It has been suggested that the bacilli are brought to these follicles by the lymph stream, which would support the contention of those who believe that intestinal tuberculosis is seldom if ever primary.

The local process begins as a small gray nodule in the submucous tissue, growing larger until the center breaks down by caseation and leaves the typical tuberculous ulcers with elevated, irregular edges. The ulcer deepens, broadens and may coalesce with others. It is usually placed transversely to the long axis of the intestine. Caseous nodules may form in the intestinal wall. The diseased process extends by the formation of small nodules in the adjacent peritoneum, and adhesions are formed which have a protective action against the spreading of peritonitis. Perforation is relatively more frequent in younger children in whom ulcers grow more rapidly. Walled-off abscess occurs in the neighborhood of the ileum, usually in the right lower quadrant of the abdomen. A small percentage of appendicitis is tuberculous, and may give chronic or recurrent symptoms, less typical than the ordinary form and may be masked by the tuberculous lesions elsewhere in the abdomen.

Stricture of the intestine results from scar formation in the wall of the ileum. This occurs in the hyperplastic type, in which there is thickening of the intestinal wall. The peritoneal cavity may be obliterated and all of



the viscera adherent from the thick exudate. The peritoneum through certain or all of its extent is studded with tubercles as the disease advances. This is often noticed on the surface of the liver and spleen. The mesenteric nodes are involved and may become cheesy.

Miliary tuberculosis is the variety characteristic of infancy, with ulcer formation in the intestinal lining.

*Peritoneal Lesions.*—In only  $2\frac{1}{2}$  per cent of cases is the disease found without older lesions elsewhere in the body.

Tuberculous nodes, tubercles on the peritoneum, and adhesions between the intestines may all be present in the same case. Wollstein found no healed lesions in 184 necropsies. The types are classified by their clinical manifestations, as they all belong to one disease, but at different stages may show an exudative tendency (ascites), due to irritation from the presence of tubercles, a dry (plastic) or nodular form. In the ascitic stage the omentum may be rolled up and lying across the abdomen. The nodular type shows marked thickening of the omentum and peritoneum, which are thickly studded with tubercles. The dry form is characterized by adhesions which represent a reparative process. Recovery is made possible by the encapsulation of the tubercles. The disease progresses by the coalescence of the caseous masses which results in abscess.

*Mesenteric Lesions.*—There is less tendency for tuberculous infection of the mesentery to spread by the blood stream. Caseation of the mesenteric glands is seldom associated with cases having miliary lesions elsewhere or with tuberculous meningitis. The disease, however, may spread by the lymphatics to neighboring organs and even through the diaphragm to the pleura. Intestinal ulcers and peritonitis may be associated. Encapsulated peritonitis occurs in older children, and can be recognized by a cyst-like tumor felt through the abdominal wall.

*Symptoms.*—Except when there are acute obstructive symptoms, the disease is usually indefinite at the onset. The condition of the child which attracts attention is the poor appetite, the presence of fever and the marked loss of weight. The child becomes pale. Sweating may be present. When the abdomen is observed, it will be found larger than normal and attempts at palpation will be resisted by the child. At a favorable time of examination, nodules which represent mesenteric glands may be felt through the abdominal wall. Tumor masses, at first suggestive of fecal concretions, are made up of enlarged glands, matted intestines or rolled-up omentum.

The development of acute tuberculous peritonitis may show a sudden filling of the abdomen with fluid. During this stage there are high fever and gastro-intestinal symptoms, consisting of vomiting, abdominal pain and distention. In the typhoid-like form the fever remains high; all the symptoms in the abdomen may be masked if there is a generalized tuberculosis. In the stage of ascites, the abdomen is greatly enlarged, the veins of the wall are prominent, and much fluid is present in the peritoneal cavity. Adhesions

form when the exudate becomes fibrinous, and the intestines become matted together or adherent to the abdominal wall. It is not at all rare for perforation to occur, and a fistulous opening empty into the umbilicus. Pus or fecal matter may be seen coming from the umbilicus or the opening in the abdominal wall. In older children the disease is more chronic. There are symptoms of ill health and indefinite abdominal pain for months or a year before the disease is proven.

Intestinal disease causes persistent *diarrhea* with stools which are somewhat loose or at times distinctly watery. *Blood* is usually present, sometimes in considerable amount. If the stools are constipated, as happens in atypical cases, the movement may be streaked with blood. The evacuation may contain pus or shreds of mucus. The number of the stools is increased by the presence of food. There is not so apt to be blood in the passages of

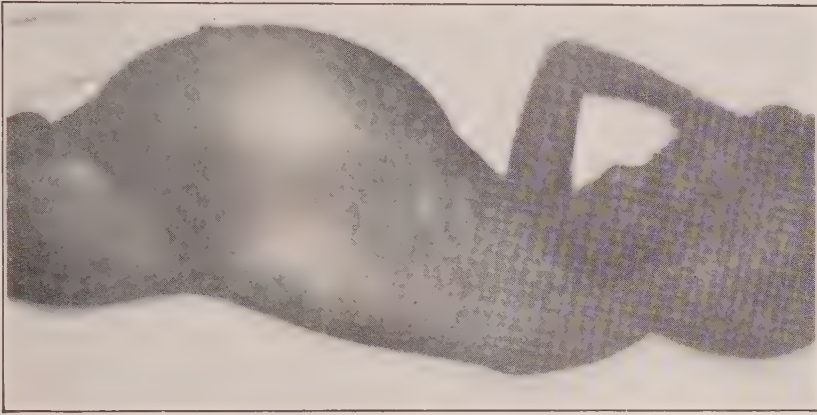


FIG. 47.—THREE-YEAR-OLD NEGRO BOY WITH ASCITIC TYPE OF TUBERCULOUS PERITONITIS

infants. Pain during straining, and abdominal tenderness in the region of the umbilicus and right lower quadrant, are evidence that the tuberculous process has invaded the serous coat of the intestine. Discomfort accompanies the diarrhea, and may be a most severe manifestation and show no tendency to subside. The amount of fever, usually higher at night, depends upon the extent and severity of the diarrhea and whether the disease is generalized. Clinically the case may resemble typhoid fever, with marked diarrhea, high fever and stupor, or dysentery, in which the bloody nature of the stools is pronounced.

Emaciation is progressive, resulting from the persistent diarrhea, the loss of fluids and blood, the lack of appetite and the presence of continued toxemia. Abdominal distention with gas indicates the involvement of the mesenteric glands, which can be felt only when deep palpation is possible. By rectal examination masses may be felt, consisting of immovable nodules lying near the spinal column.

The following is a report of the findings in peritonitis taken from the hospital records at Kansas University Hospital:

"J. H., three-year-old colored boy, has lived with mother until seven months ago when she died of pulmonary tuberculosis. Father and aunt also have tuberculosis. The boy was apparently normal until one month ago when the abdomen began to enlarge steadily. Diarrhea has never occurred but there has been occasional constipation. The appetite has always been good, but the body weight is decreasing; no night sweats nor any symptoms of meningitis have been present.

"The boy suffers no pain, but becomes out of breath on exertion. The abdomen is large and protuberant, a transmitted fluid wave is easily elicited. When he is recumbent, the summit of the protruding abdomen is tympanitic. A change of position to the lateral shows tympanites in the uppermost flank. The liver is

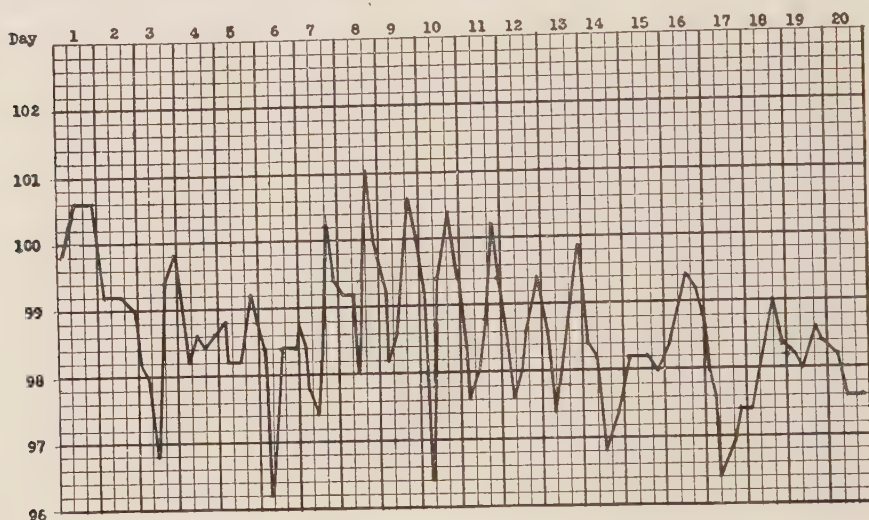


FIG. 48.—TYPICAL TEMPERATURE CURVE WITH MORNING REMISSIONS AND EVENING EXACERBATIONS IN TUBERCULOUS PERITONITIS

two finger-breadths below the costal margin. Physical signs in the chest entirely negative. The radiogram of the chest shows a moderate amount of increase in the peribronchial density of both lungs, not enough to be confirmatory of tuberculosis in that region. The right border of the heart extends well to the right of the middle line, an enlargement of the cardiac area. The tuberculin skin reaction (Von Pirquet) was hyperactive, resulting in vesicle formation and desquamation. The temperature varies from normal or subnormal in the morning to  $101^{\circ}$  at night, as shown on the specimen of graphic chart herein presented."

**Diagnosis.**—A suggestive clinical history is obtainable in many cases. In the young child the diagnosis is often missed, due to mistake of regarding emaciation as a feeding or nutritional disorder.

In generalized tuberculosis the local abdominal symptoms may stand out sufficiently to be recognized. The child should always be examined for evidence of symptoms and physical signs in the chest, followed by a roent-



gen-ray picture. When the lungs, heart, kidneys and liver are clinically normal, the chronic nature of the disease in the abdomen should easily suggest the diagnosis. The von Pirquet test may be negative early in the course, but becomes positive when the infection and caseation have become extensive.

Radiographic evidence of calcified tuberculous mesenteric glands is shown by the presence of honeycombed areas in the abdomen, in front or to either side of the vertebral column. In a series of 120 children who reacted to tuberculin tests, there were 17 per cent in whom such evidence was found, according to Dunham and Smythe.

In acute tuberculous peritonitis the symptoms are more active. Much fluid in the abdomen should always suggest tuberculous infection. Congenital syphilis is somewhat similar in the wasting infant, but there is no ascites.

The presence of nodular masses on palpation of the abdomen should add to the diagnostic evidence of mesenteric tuberculosis. The glands, however, may not be large enough to be easily palpated. Tuberculous mesenteric glands cause more marked toxemia and more severe nutritional disturbances than tuberculous infection of the other glands of the body.

Diarrhea alone does not prove that the tuberculous child has intestinal ulcerations, for acute indigestion is a frequent occurrence in children who have any severe infection outside as well as within the intestinal tract. Digestive upsets are particularly liable to happen, and in abdominal tuberculosis, foods containing large amounts of fat are not well tolerated. The diagnosis is difficult in early childhood at which time dysentery and nutritional disturbances are common.

In a child under two years of age, a positive tuberculin test is diagnostic for tuberculous infection but does not enable one to say that the disease is active, or to localize the bodily region involved.

Ulceration and stricture of the intestine may be shown by roentgen barium pictures and will help in the diagnosis of those cases which have chronic diarrhea, abdominal pain and enlargement, tenderness, rigidity and obstruction.

The presence of tubercle bacilli in the stools is a valuable aid if not due to the passage of swallowed sputum, in which case they indicate pulmonary tuberculosis.

The existence of suggestive history, either personal or familial, the presence of a positive tuberculin test in the young child, the evidence of tuberculosis elsewhere in the body, the persistence of a slowly developing diarrhea accompanied by bleeding, the recognition of mesenteric nodules and of an enlarging abdomen with fluid, and the progressive emaciation, make probable the diagnosis of abdominal tuberculosis.

**Complications.**—Intractable diarrhea and marked ulceration are evidences of severe progress of the lesions. The occurrence of pronounced



caseation, extension of the disease to new areas in the peritoneum, formation of abscess, obstruction from the presence of masses of enlarged glands or from ileus, are complications which may be expected.

**Prognosis.**—In a previously vigorous child, the uncomplicated case of peritonitis tends to final recovery after six months or a year. The natural tendency of the tuberculous process is for a limitation of extension by shutting off of the blood supply to the part, and encapsulation with a fibrous formation. In the favorable case active absorption will take place. In coincident pulmonary disease or with preceding feebleness the child's chances for recovery are much less. When the intestines are matted together, the course is chronic but the outcome still good if the viscera are left alone. Low body temperatures especially for a portion of the day give some relief if the other symptoms are not active, for the child will temporarily have a better appetite. High fever indicates the presence of a complication. Diarrhea usually means the existence of intestinal ulceration. Other unfavorable signs are intestinal paresis and distention, obstruction, rapid emaciation, suppuration; rupture of an abscess and general spread to the lungs and meninges make the prognosis unfavorable, for simple laparotomy cannot cure the disease. The best results which I have seen reported were in twenty-seven cases treated medically, of which twenty-two recovered.

Tuberculous ulceration of the intestine is extremely chronic in older children, but at any age shows little tendency to recover. Death seldom occurs from ulceration alone, but is due to the involvement of the neighboring organs and to the previous or subsequent infection of more vital regions. The infant usually dies, but the older child passes into an asthenic and emaciated state, with a persistence of the intestinal disease. It has been suggested that surgical intervention might be of benefit if the ileum could be excised or put at rest by a short-circuiting anastomosis. The fatal course is one of persistent toxemia, hemorrhage, and not infrequently perforation, peritonitis and sepsis. Death occurs within two to four weeks after the appearance of these serious complications.

**Treatment.**—*Prevention.*—The general practitioner should be actively concerned in the prophylaxis of tuberculosis. The child should not be exposed to the environment of tuberculous individuals. The infected parent is a positive menace to the young child from its very first day of life. One of the recent cases of generalized tuberculosis which I happened to see was a breast-fed infant who became infected by the presence of a tuberculous visitor in the home for one day only.

The difficulty of obtaining safe milk is in many communities very great, and can be solved only by the rigid exclusion of all tuberculous cows and employees from the dairy. This is a public health problem and requires constant vigilance. The alternative is the boiling of all milk fed to children. This is thoroughly practical for the child under five years of age. Older children object to the taste of boiled milk, but they should be taught

to drink it if milk of unproven purity is to be used. In such instances milk may be safely cooked in food. In Scotland, the students of the milk supply have recently recommended the sterilization of all milk and bottles, sold with sealed caps as a method of reducing the incidence of tuberculosis.

Recent reports from France give some hope of the benefit from prophylactic treatment consisting of vaccine made from attenuated cultures of tubercle bacilli. Further results from the use of this method will be of great interest.

**General Treatment.**—Complete rest in bed and in the warm sunshine will do more than all other measures except proper feeding. The child may have the bed moved out of doors and spend much of the time there. In the winter months, daily exposure to the ultraviolet ray has proven of marked value in mesenteric and peritoneal involvement. The family should secure a quartz lamp by rental or otherwise so that the treatment may be given at home, or in the hospital, without causing fatigue of the child.

Adequate food is needed. Easily digestible and varied articles of diet should be provided, and the appetite for these stimulated by iron, arsenic and small doses of nux vomica. Once a week a blood transfusion is advisable in cases of repeated or severe hemorrhage. Intravenous injection of 5 c.c. of a 5 per cent calcium chlorid solution has been attended with good results as regards the bleeding.

**Treatment of Special Symptoms.**—*Pain* is not so marked when the child is quiet in bed, but it is severe if acute sepsis develops. Local application of hot stupes relieves mild pain and tenderness, but opium should be used when pain is severe.

*Vomiting* is mild unless there is intestinal obstruction, which should be treated surgically. Lavage of the stomach with soda bicarbonate solution followed by bismuth powders will be of help in vomiting.

*Diarrhea* may be steady and severe, requiring opium and bismuth to check the peristalsis. However, watery stools if not too copious and exhausting may be of value in depleting the abdominal fluid in the case of ascites. For ulceration in the rectum or colon, hot-water enemata once daily may reduce the number of stools, but as the disease is usually in the ileum and cecum, little may be expected from high injections.

*Constipation* may be present where there is marked ascites. High enemata are indicated. Saline laxatives may reduce the amount of abdominal fluid but should not be used if there is ulceration.

*Scanty Urine.*—The urine is scanty when the fever is high; or much fluid is being lost by way of the stools. The excretion may be stimulated by the administration of much water and other liquids by the mouth.

*Flatulence.*—The discomfort from gas may be relieved by the colon tube. The diet should be simple but nutritious, not necessarily liquid. Buttermilk is better tolerated than sweet milk; starches should be temporarily discontinued.

**Surgical Treatment.**—The present opinion of operative interference is somewhat different from that of a few years ago. The best method is generally to let the abdomen alone and use only medical and hygienic treatment. First of all is the question of emergencies. A sudden extensive increase in fluid, which causes sufficient distention to produce extreme dyspnea, may be temporarily benefited by removal of some of the fluid, but it soon recurs. Tapping is somewhat dangerous because of the possibility of puncturing an adhered intestine and the production of peritoneal infection or a fecal fistula. The adhesive case causing obstruction needs surgical interference, but in the presence of caseation may result in perforation if the operation be not carefully handled. Caseous lymph-nodes should be removed if possible.

Cyst-like abscesses should be drained by skillful surgical procedure. After drainage, cases have been reported recovering without further interference in the health. In other cases a draining abscess may become infected with pyogenic organisms and result in sepsis or a chronic fecal fistula. Collapse may follow extensive operation and handling of the viscera.

There has been much speculation as to the reason for the apparent improvement following simple laparotomy, and earlier it was regarded as due to the introduction of air into the peritoneal cavity. Air or oxygen injection has been used by some advocates for the last twenty years.

The nodular stage (mesenteric lymph-node enlargement) is not successfully treated by operation, but if the infection be localized to a small area, the surgical removal may result in a prevention of dissemination. Dry cases recover as well without operation. About 50 per cent of ascitic cases recover after the abdomen is opened, but it is difficult to prove that operation plays any part in cure, for medically treated cases do as well.

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## CHAPTER XXIII

### ABNORMALITIES AND DISEASES OF THE RECTUM AND ANUS

The three important disturbances of the rectum in childhood are congenital obstruction of the anus and rectum, prolapse and proctitis. Hemorrhoids which are so common and troublesome in later life are in children infrequent and cause little disturbance.

Rectal examination is not so commonly done as in adults, because of the usual lack of indication therefor, but it is necessary and of great value not only in the diseases under consideration, but for its general diagnostic assistance. Digital examination should be especially familiar to all who practice pediatrics. It is not only harmless but is easily performed, and the index finger may be employed in children of any age. The examiner stands to one side or at the foot of the child, who is placed upon the bed, table, or in case of younger children across the mother's lap. The finger, covered by the rubber glove, should be well lubricated, gently and slowly introduced through the sphincter. The shallowness of the pelvis permits the finger to reach well above it and to be swept around in all directions. The opposite hand of the examiner enables one to palpate or define any intervening tumefactions. A trace of blood sometimes follows the finger when it is withdrawn, due to rapid stretching of the sphincter.

Digital examination of the rectum is of use in disclosing such inflammatory masses as may be found in appendicitis, abscess and intussusception, also fecal impaction, tuberculous lymph-nodes and sarcoma in the lower half of the abdomen.

**Congenital Abnormalities of the Rectum and Anus.**—The imperfect development of the rectum and anus is found in one out of probably ten thousand newly born infants. One can say that the general practitioner may never have a case in his own practice, or at most not over one or two. The reports are those based upon death statistics and do not represent the total number of cases, as many are saved by simple and skillful operative measures. Our only reliable information comes from the few published records of large obstetrical clinics. Quinland collected from the museum of the Harvard Medical School, twenty-seven cases of atresia of the intestine and imperforate anus. In some of his cases the associated intestinal defect was as high as the small gut. One case had a history of a similar defect in the family.

**Embryology.**—Three types of imperfection of this region are found and these may exist singly or be combined in various ways.



Simple imperforate anus has a normal rectum, but no external opening. The site of the anus is replaced by skin, or there may be a superficial opening which ends in a mucous septum or a shallow blind pouch. The sphincter muscles are present. The presence of a single defect of this kind is less serious for the child. However, the abnormalities of the anus are more apt to be associated with other defects of the intestinal tract. The one which may be expected is an atresia of the rectum with one to three inches undeveloped. This second variety then has an incomplete rectum, with or without an imperforate anus.

The third type is more serious and often complicates the other two varieties. The rectum and bladder may be joined as one common receptacle, or by the persistence of this embryological state to a less extent, the rectum communicates by a cloacal duct with the bladder, urethra or vagina. Fistulous openings empty the intestinal contents into these regions.

The deformities are due to some accident in the evolution of the cloaca. The dilated lower end of the rectum in early fetal life, from which develops the primitive bladder, fails to subdivide into bladder, urethra and vagina, and these organs remain in communication with the rectum, usually through a cloacal duct. The imperfection of the anus is the result of another anomaly in development whereby the anal membrane which is derived from the cloaca fails to rupture and form the anus. The rectum does not develop sufficiently to unite with the anal segment.

The earliest symptom is the absence of meconium stools. The symptoms vary somewhat, depending upon the presence or absence of a fistulous opening and discharge of intestinal contents through the bladder, urethra or vagina. When no such communication exists the symptoms are those of a complete intestinal obstruction.

Observation during bathing the newly born infant will disclose the complete absence of an anal opening, or in case of imperforate anal canal the discovery is made when attempting to take the rectal temperature or give an enema because of the lack of a stool. Usually a normal open anus is evidence that no rectal atresia is present, but does not preclude the possibility of obstruction in the rectum or higher in the intestine.

Sooner or later it is discovered that meconium or feces is passing from the vagina or in the urine. The location of the fistula in the bladder is suggested by the constant presence of feces in the urine. Urethral fistula also permits of contamination of the urine, but the fecal matter may be voided independently of urination.

Vomiting occurs in all cases of complete obstruction, but may not begin for two or three days. It is most severe when there is an associated defect in the upper intestinal tract.

The abdomen becomes distended and tympanitic and the superficial veins of the abdominal wall are greatly enlarged.

The contamination of the bladder soon causes grave complications, with

cystitis, dilatation of the ureter, pyelitis, the ascending type of nephritis, and sepsis. Urethral fistula is likewise a menace to the bladder and upper urinary tract, but infection is slower in developing.

Death occurs in five or six days if the intestinal obstruction is complete.

*Treatment.*—Simple cases should be carefully punctured by knife or scissors, avoiding injury to the peritoneum. When a fistula is found in the vagina, an instrument may be passed through it down to the anal membrane and the membrane opened.

If the imperforate anus be complicated by a defect in the continuity of the rectum, an operation through the perineum is regarded as the simplest for uniting the end of the intestine to the anus. When the rectum and bladder communicate with each other, an immediate colostomy is advisable for drainage of the feces and preventing of urinary infection. Bevan recommends the creation of this abdominal artificial anus by which the

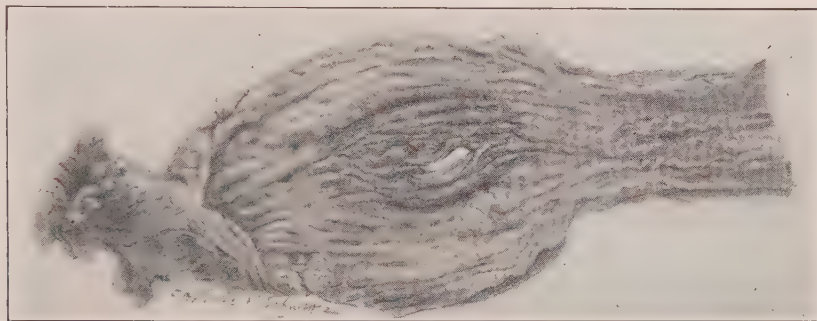


FIG. 49.—IN THE CENTER OF THIS DRAWING A PERFORATION OF THE RECTAL WALL IS SEEN

This accident occurred in a newly born infant, causing death from peritonitis. The perforation had been produced in the attempt to pass a bougie through an atresia of the anus.

fecal discharges are diverted from the urinary tract. Later if found by the outline of the barium shadow that the communication between the rectum and bladder is not too extensive, it may be possible and advisable to do a secondary operation for separation of these organs. The operation is also indicated in urethral fistula.

Disasters are common from operation for any of the more extensive types of defects. Hemorrhage, infection of the peritoneum, cellular tissue and kidneys are postoperative complications, with sepsis and terminal pneumonia.

**Prolapse of the Rectum and Anus.**—The occurrence of rectal prolapse is found to be more common at the age of two or three years. It is often seen during illness, but in some cases the child may have been entirely well. The exciting factor is usually diarrhea or constipation, during which severe straining is produced. Acute diarrhea with almost constant stools is most apt to cause prolapse, due to the marked increase in peristalsis, the rectal tenesmus, and the inflammation of the colonic and anal mucosa. Pin-worms may cause it, due to the irritation and straining which are an accompaniment.

Cases are reported from the tenesmus following stone in the bladder, but this is a rare condition in childhood.

There is some difference in opinion as to the underlying causes. The investigations of Todd show that infants normally have no greater tendency than adults to a laxity of the rectosacral attachments. In the developed case, however, there is obviously insufficient support for the rectum. Exhausting illness and the disappearance of muscle tone and supporting fat render the child susceptible to prolapse.

No disagreement exists as to the predisposing influence of anatomical peculiarities in the shape of the pelvis and the direction of the rectum found in childhood. The straighter sacrum in early life is in marked contrast to the hollow sacrum of the adult. The rectum is thereby directed more vertically. It lies against the straight sacrum without the protection which the hollow pocket permits in later life. The small shallow pelvis also is a predisposing influence. The higher position of the bladder and uterus permits of more pressure upon the rectum.



FIG. 50.—PROLAPSE OF THE RECTUM

The prolapse may begin at three levels. When it originates high up in the pelvic colon, the mass does not protrude from the anus. Another type begins at some distance above the anus and will be protruded. Both of these forms are really invaginations. The true type of rectal prolapse starts from the margin of the anus, and a ring of inflamed mucosa is pushed through the anal opening during the act of straining. The most common type is incomplete, in which only the anal mucous membrane protrudes. In a severe degree, the entire wall of the rectum comes down, and several inches

including the sigmoid may protrude and remain for sometime unreduced.

*Symptoms.*—In mild cases the mucosa appears only with the stool, and then spontaneously returns. As the tendency increases the anal sphincters become much relaxed and the bowel may protrude independently of defecation, even while the child is playing. A mass of various size is found, the surface of which is dark red and bleeds easily. The intestine comes down by the effort of straining, crying or coughing, and in marked cases it may only be reduced with difficulty, due to the swelling and constriction at the anal outlet. The mass may remain outside the anus until ulceration or inflammation occurs. In such a case the possibility of a true intussusception should not be overlooked, though such seldom protrudes. Under proper care the tendency to recur decreases as the child gets older.



*Treatment.*—The physician is usually summoned when the prolapse is down. The immediate treatment is the reduction of the bowel by grasping the tumor and gently pressing against the mass, while the finger, well covered with vaselin, enters the lumen of the rectum until the replacement is complete. A firm compress is then applied to the anus and held in place by a four-tailed perineal bandage. The thighs are bandaged together or the buttocks strapped with adhesive plaster. The child should be kept lying on the abdomen for one week, with intervals of rest in the dorsal position. For two weeks longer the confinement to bed is advisable.

The after treatment is primarily the avoidance of further straining. In the beginning of the curative effort, the bowels should be kept open by a gentle enema with cold water, the child placed on the bed-pan. At all times the child should not be on the feet during defecation. Constipation is prevented by a diet which keeps the stools soft, and if necessary a daily dose of mineral oil and agar. Diarrhea is likewise to be avoided.

Improvement in the body tone and the development of plenty of fat are important factors in most cases. Strychnin, grain 1:200 in infancy and grain 1:100 at two or three years of age, given in simple elixir, three times daily, is of value.

Prolapse in infancy tends to a short duration and spontaneous disappearance. The severer cases usually get well under proper treatment within three weeks.

Radical or surgical measures have in my experience never been necessary. Some men of large experience have advocated the injection method or operation. In one the effort is made to produce adhesions between the rectal wall and the surrounding tissues by the injection of 1.5 c.c. of pure alcohol under a general anesthetic. The needle is introduced at each side of the anus, the finger in the rectum guarding against entering the gut. Twenty-six cases thus treated have all been cured. The surgical treatment consists of fixation of the posterior rectal wall to the sacrum by silk suture. This method is reported by Petrén as successful in the fifty or more cases thus treated. All of these vigorous procedures are followed by rest in bed for several weeks, and the avoidance of straining.

**Proctitis.**—Inflammation of the lining of the rectum is not unusual in children and there are numerous causes for it. Proctitis may exist as a separate entity, due to local irritative factors both mechanical and infectious. The best-known example of catarrhal proctitis is that produced by pinworms. Pustules near the anus and infection of the anal canal may cause the spreading of inflammation to the rectal mucosa. Foreign bodies which have been swallowed or introduced through the rectum may cause mechanical irritation. The use of suppositories or frequent enemata has been regarded as irritating to the mucosa, but it is doubtful if inflammation is ever produced in this way when care and gentleness accompany the pro-



cedure. The ill-advised addition of strong soaps and corrosive drugs to irrigating solutions can undoubtedly be harmful.

As a part of a general involvement of the large intestine, ulcerative proctitis may be associated with dysentery, or a chronic ulcerative colitis.

The symptoms of acute proctitis are frequent straining, pain on defecation, mucous and bloody discharge, and evidences of redness and inflammation around the anal orifice. There is not infrequently a prolapse of the anal mucous membrane. Frequent urination results from associated irritability of the bladder. The course depends upon the etiology.

The treatment lies in the removal of the irritative cause. The child should be kept quiet in bed and no laxatives given. Local measures consist of gentle irrigation with hot water and the instillation of olive oil.

**Gonorrheal Proctitis.**—When blood, pus and mucus are discharged from the rectum of a child without definite diarrhea or pin-worms, the source may be a gonorrheal infection and inflammation of the anal and rectal mucosa.

There are several ways in which such infection occurs. One is by the thermometer used in children's wards. Institutional epidemics of proctitis and vaginitis have been produced in this manner. In a hospital ward six boy infants were infected by the thermometer. The infection may be carried in the wash room of the hospital. I have seen one case of proctitis develop from the child's profuse vaginal discharge which constantly bathed the perineum and anus with pus. That more complications of this kind do not occur is only to be explained upon the higher resistance which the rectum has for the gonococcus. Contamination of the anus at birth from the discharges of the mother may go on to vaginal and rectal infection.

Criminal assault is the cause of infection in probably more cases than we know, especially in young children who are unable to report it. Pollack called attention to the large number of little children in a large city who had been attacked and infected, and during her study found seven cases involving the rectum.

The stools first show mucus and then become loose, green, bloody and purulent. The lining of the rectum is reddened, ulcerated and may become prolapsed. There is little fever. The examination of smears taken from the rectum shows the gonococcus.

Complications are not uncommon. Ischiorectal abscess may occur, and a complication with vaginitis, urethritis and bubo. As a result of metastasis through the blood stream, arthritis and endocarditis are sometimes caused.

*Treatment.*—The irrigation with silver nitrate, 1 : 3000 solution, alternating with acriflavin, 1 : 1000 solution, is the best treatment in the early stage of the infection. Long-continued infection cannot be cured.

If rectal temperatures are taken, prophylaxis should be attempted by the use of individual, well-cleaned thermometers. No case of vaginitis or specific proctitis should be kept in proximity to other children, nor should the

same kitchen, bathroom or nursing service be used. From the private practice of many years with routine rectal temperature-taking, I have not known of accidental infection, but it is better to discontinue this method in girls with vaginitis, or if there be any carelessness in the cleansing and indiscriminate use of the clinical thermometer. Dispensaries will do well to exercise the greatest care in their methods of disinfection.

**Fissure of the Anus and Rectum.**—Fissure is one of the common, less important, but often disturbing rectal disorders of young children. It is a frequent reason for consulting the physician. That such a small lesion can at times be productive of so much disturbance would seem improbable in comparison with the other gross lesions which we have just considered. But the anal region is well supplied with nerves, pain is often extreme, and after much experience with young children one reaches the conclusion that they may be markedly affected by seemingly trivial annoyances.

The history is usually one of recurrent blood-streaked stools. Fissure occurs most usually in the infant who has the hard dry stool of the fat-constipation type. The passage of the movement causes an overstretching of the anus. However, the lesions can occur in diarrhea and from the irritation of pin-worms. In the latter eczema of the anal cutaneous surface may have been caused by scratching and a fissure produced in this way. The rough use of irrigating nozzles or non-lubricated thermometers may cause abrasions of the anal mucosa.

The fissure may be seen at the mucocutaneous juncture. When more deeply situated, it is necessary to stretch the sphincter in order to inspect the anal canal. Lesions beyond this site, or in the rectum, can only be made visible by the use of a small (nasal) speculum. The lesion is most often on the posterior wall, but it may be hidden by the mucous membrane of a complicating prolapse. The bleeding from fissure is slight, and is of minor importance as a symptom, compared with the spasm of the sphincter. This is accompanied by pain and screaming, and an avoidance of defecation, so that there is further drying out of the retained feces. The general health of the child in some instances suffers from the discomfort, the restlessness and lack of sleep. Retention of the urine is not an infrequent accompaniment of fissure.

Fissure is sometimes complicated by infection and an abscess may form in the submucous or ischiorectal region. This usually is the result of further injury by neglect in securing soft easily voided stools, or from lack of cleanliness.

*Treatment.*—Hot towels applied to the perineum or frequent immersion of the child in a warm bath are of help in relieving the anal spasm. When this is severe the stretching of the anus may be necessary, and it is considered curative. Gentle injection of warm boric acid solution is an advisable procedure for a few days. In severe cases with ulceration, a wick covered with balsam Peru is inserted into the anus. The constant attention

to the bowels is a necessity, and for this purpose mineral oil and agar and a laxative diet are the most important. The cure is prompt and only in rare and neglected cases do complications occur. Deeper ulceration of the rectum may need the application of silver nitrate or cautery.

**Hemorrhoids.**—Piles are infrequent during childhood and they are present for only a short time. Children brought with the diagnosis of piles will usually be found to have a small anal prolapse or a redundant cutaneous or mucous tag, near the anus. Disturbances of the lower bowel such as straining from diarrhea or constipation are the exciting factors. The lesion is usually external and small. Pain and blood may accompany defecation. In case of inflammation the child should be put to bed. It has never been necessary in my experience to submit the child to operation. Stretching of the sphincter may be helpful. The correction of intestinal disturbances, the avoidance of constipation by diet and cold-water enema, and finally the establishment of daily regular habits of defecation are usually sufficient. The symptoms and local treatment are not different from those of adult life.

**Polypus.**—Recurrent hemorrhage is the most characteristic symptom of rectal polyp. Bleeding is frequent and the amount passed considerable. Unless the polypus protrudes or is passed through the anus, it is not usually diagnosed until all other sources of blood are excluded and a digital or proctoscopic examination is made. Fissure is the principal lesion in childhood with which it may be confused, but is more common and the amount of blood less. Some pain may accompany defecation. Hemorrhage is due to ulceration or to the separation of the vascular growth from the rectal wall. The growth may be single or multiple, the latter consisting of numerous growths varying from the size of a millet seed to a tumor an inch in diameter. A polypus may have either a broad base or a more or less narrow pedicle by which it is attached to the gut. Large growths are uncommon in childhood, but it is probable that multiple small polypi are responsible for many of the minor hemorrhages which parents report. The situation may involve not only the rectum but the colon as well.

Spontaneous evacuation of a polyp is a common termination, while others of a more fibrous consistency necessitate operative removal by ligation or excision.

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## CHAPTER XXIV

### INTESTINAL PARASITES

The possibility of intestinal infestation with parasites should not be overlooked. The presence of worms indicates that the child is being deprived of nourishment needed for human use. Severe and even fatal results have been known to occur from the migration of certain types, or from their presence in large numbers. Even though, in most cases, there may be no symptoms or bad effects, these are possible, and the harboring of any parasite is, to say the least, loathsome.

The subject of worms is of great importance and concern to the average mother and other adult members of the household, who may even reach a state which might be termed parasitophobia. No indisposition is too trivial or too severe to escape an assumption of a parasitic cause. I have seen a case of acute infantile paralysis neglected for several days on this basis.

Not infrequently a mother brings in a specimen of round-worms with the statement that her physician had always laughed at the suggestion that her child might be infested. This tendency to skepticism on the part of the medical profession is often unwarranted, and is partly responsible for the lay practice of patronizing advertised nostrums which have a large sale. It is common for such a medicine to be given as a routine whenever the child complains.

The skeptical view held by so many physicians is probably due to the infrequent examination for ova in the stools, and the unknown incidence of parasitism in the children of the community. Usually the passing of worms is the first and often the only symptom. Remembering the persistence of the infestation, a diagnosis is warranted on the positive earlier history alone. The physician, however, will do well to make a complete physical examination of the child in order to discover all possible causes for ill health, but he should search for evidence of parasites and ova in the stools. The technic of fecal examination for this purpose is not difficult. Infestation with a single variety is the rule, but some children have more than one type. The increase in the number of eosinophils in the blood count makes the diagnosis more certain, but cases of positive though mild infestation may not always have eosinophilia.

Infection more easily occurs in the institution or school, where it is spread by contamination in the usual ways. The occurrence is more common in homes and institutions that are not kept clean, the hygiene of the



children neglected and association with dogs frequent. Infants are not so much exposed and are seldom infected before the runabout age.

Few general surveys have been made in this country. The studies made in institutions are not indicative of the general population, since such children are poor, previously neglected and obviously more apt to be infested. Under sanitary conditions the incidence of ova in the stools is probably under 18 per cent, but in institutions where children are not periodically examined and their hygiene not directed, the incidence may reach 80 or 90 per cent.

Symptoms are as commonly ascribed to uninfected children as to those with parasites demonstrated in the stools. It has been my experience, however, to be able to elicit a history of rather indefinite symptoms which should suggest parasites when other diseases have been excluded.

Substances resembling parasites and their eggs are often difficult to distinguish. One should inquire into the nature of the recent food, which leaves a residue in the stools mistaken by the mother for worms. Children ingest innumerable foreign articles, such as wood fiber, stems of plants, strings, thread, etc., while many of the fruits and vegetables leave some of their fibers and pulp which are macroscopically and sometimes microscopically not easy to exclude from varieties of parasites. Orange and lemon pulp may resemble flukes, banana fiber suggests the dwarf tapeworm, celery and rhubarb leave stringlike fibers not unlike thread-worms, and onion laminae the segments of tapeworm. More difficult is the differentiation of yeast and other vegetable cells, spores and pollen, from the ova of parasites.

**Varieties.**—The most common animal parasites found in practice in this country are the pin-worm and round-worm. Another variety of the nematodes, the whip-worm, furnishes the greatest incidence of eggs found in the feces, but the worm itself is seldom seen. The hookworm, the fourth common member of the nematoda group, can cause the most serious general disturbances and is widely distributed and endemic in certain of the southern states.

In children the dwarf tapeworms are more common. Seldom does one see the large type.

The lower forms of animal intestinal parasites infest the alimentary canal, and are sometimes found in children. They belong to the order of protozoa, the most important from a pathogenic standpoint being the entameba. The writer has seen fit to place amebic infection in this chapter, rather than in the group of dysenteric diseases, where, however, it equally well belongs. Two other types of protozoan infestation will also be mentioned. These are the *Lamblia intestinalis* and the *Balantidium coli*.

**Oxyuris vermicularis (Pin-Worm, Seat-Worm).**—Pin-worms will be discovered by the observing mother or attendant, though vegetable or fruit fibers are often mistaken for parasites. The worms are found moving on the surface of the stool or in the abundant mucus which is passed. In

small children who are not carefully cleansed after a bowel movement, the worms may be found some distance from the anus, particularly in the vulvar region and around the buttocks.

The individual parasite is white, thread-like in appearance, but tapering at both ends. Its length is from  $\frac{1}{4}$  to  $\frac{1}{2}$  inch. Eggs are not always easy to find. They are microscopical in size, about  $\frac{1}{500}$  inch in diameter, somewhat oval, but irregular in outline.

It cannot be said that there is any year of age in which this form of parasite is most common. In institutions for children in one American city, it was found in 7 per cent of individuals. That they are fairly common in England is shown by the figures given by Still who found them in one-fifth of a large series of autopsies in all children under twelve years of age. In his cases worms were usually present in the appendix; in some they were exclusively in that location. The cecum, colon and rectum, however, are the usual seat of the infestation. The numbers may be uncountable.

The original mode of entry is by the mouth. Flies may transmit the eggs from stools to exposed food. The most common carrier is the hand of the infested child, by which playthings, furniture, fruits, dust and probably many other things become contaminated and reach the hands and mouths of other children. The common use of the handkerchief and towel is a source of spread. Infested children will at times have ova under the finger nails from scratching the anus and from lack of cleanliness in the toilet. The common practice of picking the nose may carry ova to the nostril, where they may be found in the secretions.

As regards the habitat, it is generally considered that the stomach and small intestine are the breeding places for the oxyuris. Formerly it was thought that ova do not mature until reintroduced into the mouth and passed through the stomach. Pin-worms have been found in the stomach. Undoubtedly eggs are carried by the child to the mouth and reinfection caused. The worms are present in large numbers in the large intestine, being demonstrable in the rectum. They may reside in the appendix not only of children but of adults. The finding of young and immature worms in the appendix would suggest that it is one of the hatching places. The persistence of this parasitic infestation is explained best by the inaccessibility of the appendix and diverticulum for the action of vermifuge, and by the re-ingestion of ova from contaminated articles reaching the mouth. The oxyuris dwell in the copious mucus which they stimulate by irritating the mucous lining of the intestinal cavity. After their passage from the anus, they may infest the vagina or preputial opening. They may crawl back through the anal orifice.

*Symptoms.*—The presence of worms in the region of the anus is characteristic of the disease. They pass either with the stool or migrate from the orifice, especially at night. The most frequent disturbances are local.

Their invasion of the anus causes severe itching which is most noticeable after the child has gone to bed. The anus is inflamed, and may become prolapsed. Abnormal amounts of mucus are secreted by the colon and rectum which show a catarrhal inflammation. The spread of the worms to the vagina and prepuce causes vulvovaginitis and balanitis.

Abdominal pain is complained of by some children, and is colicky in nature, due to the stimulation of peristaltic contractions. Secondary symptoms are bed wetting and a frequent desire to urinate during the day, due to the irritation in the rectum near the bladder.

The catarrhal inflammation may interfere with absorption of nutritive material, and produce thereby progressive loss of weight. Anemia develops in such cases. The child may vomit easily.

Nervous symptoms of lesser or greater degree are present. Undoubtedly the rest and sleep are disturbed. Grinding of the teeth is an evidence of restless sleep. As a result of the scratching, bad habits of handling the genitals and even masturbation may be formed.

Children who are susceptible to nervous influences may have periods of fright and morning headache. Habit spasm in such individuals may disappear after the oxyuris infection has been eradicated. Strabismus in rare instances may be excited by intestinal parasites. Convulsions probably are sometimes caused in this type of child. Holt and Howland mention a case of chorea caused by pin-worms. Picking the nose is a nervous habit, though as we have seen there may be a local cause for the practice.

The ova are not abundant, but the search for them in the stools is not necessary, for the oxyuris themselves are usually visible. The mucus passed by the bowel or a small portion of the stool is smeared on a slide, and a thin film examined under the microscope. No preliminary purgation or vermifuge is needed unless the search is unsuccessful.

The finger-nail test is made by wiping the dirty finger nails of the child with a pledget of cotton wet in a 1 per cent solution of sodium hydrate. The cotton is held with a pair of forceps, squeezed into a small amount of the solution in a tube, centrifuged and the sediment examined for ova. Nasal mucus also may be examined in this way. This indirect method may be tried when ova are not found in the fecal mucus.

*Complications.*—Evidence is rapidly accumulating of the frequent occurrence of appendicitis from oxyuris infestation. Series of cases have recently been reported in both adults and children. One should therefore inquire carefully into the history of parasites, and remember the possibility of such a surgical complication.

*Treatment.*—Of first importance is the use of vermifuges by the mouth. In this way only can the primary location of the parasites in the small intestines and cecum be reached. Two remedies are especially of value. Santonin and calomel,  $\frac{1}{2}$  grain each, or double this amount for older children, are given on an empty stomach at bedtime every two weeks. The



following morning a tablespoonful of milk of magnesia or 50 per cent solution of magnesium sulphate should be administered two hours before eating. Larger doses of santonin recommended by some writers may cause toxic symptoms and a transient change in the color of the urine.

Chloroform is an efficient remedy for freeing the intestines of parasites, given in the following dosage: under five years of age, 15 drops (1 c.c.); above five years, 30 drops (2 c.c.); in from 1 teaspoonful (4 c.c.) to 1 tablespoonful (15 c.c.) of castor oil, which prevents irritation from the chloroform. It is repeated once each two weeks. In cases which are resistant to other treatment and which may have a focus in the appendix, chloroform is especially useful. Tea made from garlic is sometimes of benefit in rebellious cases. Oil of chenopodium is highly recommended, but poisoning has been reported from its use.

Early and mild infestation is easily cured by persistent treatment. Chronic and extensive involvement of the intestinal tract is not so successfully handled.

Thorough irrigation of the rectum and colon will be of great benefit in washing out the parasites and their ova from these regions. It is probable that few cases are treated for a sufficiently long time, or at regular intervals. It is obvious that enemata do not reach into the small intestine or the appendix. The technic is as follows: once daily the mucus is removed by injection of warm borax solution, a teaspoonful to each pint of water. This is followed by a second irrigation with a pint or more of infusion of garlic or quassia. Another solution which is sometimes recommended is a half pint of 1:10,000 bichlorid of mercury. I have sometimes employed it, but I never feel easy for fear of some accident attending it, such as absorption or retention.

It is preferable to use drugs which are free from all danger and which are not irritating to an already inflamed bowel. All local treatment should be accompanied by repeated oral administration of vermifuge as above suggested. Persistent treatment will result in success in most cases.

Personal cleanliness is necessary. The contaminated anus, buttocks and genitals should be washed with soap and hot water after each defecation. The anus should be covered with unguentum hydrargyri after each movement and at night, which will relieve the itching and prevent the worms from migrating or reëntering the bowel. The fingers should be well scrubbed with a wash cloth and soap.

**Ascaris lumbricoides (Round-Worm).**—This variety of parasite is somewhat more common in the runabout or preschool child than in the ages from six to twelve years. Adults and infants are rarely infested. It is difficult to learn the incidence of this type of parasitism, but it is less frequent than pin-worms and the dwarf tapeworm, and more common in children than the large tapeworm. DeBuys and Dwyer found 15 per cent of infestation in children of institutions surveyed by them. The migration



from the intestine is usually the means of their discovery. Although seldom more than one is passed with a stool, there are usually others in the intestinal canal. They may be few in number, but whole masses and coils of worms have occasionally been discovered at operation or autopsy.

The child is greatly frightened at the discovery of the worm. One youngster while walking into my office vomited a long round-worm upon the carpet, greatly to her consternation and to the surprise of others. However, vomiting of a worm is not rare, and the ascaris has also the faculty of crawling up into the pharynx. When it enters the air passages, asphyxia is caused. The round-worm may migrate to the pancreatic duct, and to the liver through the bile ducts. Abscesses and peritonitis have been reported from their perforation of the intestines. A worm may pass unassisted through the anus.

More than one child in the family may be infected by the carrying of the ova from one individual to another.

Infestation occurs through the mouth from the eggs which are carried upon the fingers or in water or raw food which has been contaminated with soil or excrement. Lower animals are infested. The cycle of development of the parasite is not understood, there being a difference of opinion as to where the maturing and hatching of the eggs take place. The worms dwell chiefly in the small intestine where the ova are deposited.

This ascaris is named "lumbricoides" because of the resemblance in shape and size to the common earth-worm (*Lumbricus terrestris*), the latter, however, not being a parasite. The round-worm is easily recognized. It is long and cylindrical with pointed ends. The size varies from 4 to 12 inches in length (10 to 31 cm.), and the width from  $\frac{1}{8}$  to  $\frac{1}{4}$  inch (0.3 to 0.6 cm.). The color is white or bile-stained, and the surface marked by transverse striations. The eggs are present in large numbers in the feces, showing a marked difference from the scarcity of oxyuris ova. The ova are yellowish brown, oval and microscopical in size (about  $\frac{1}{400}$  inch in diameter).

The female is about twice the length of the male. The sex can also be determined by the curved and bristled end of the tail in the male, and the straight conical tail of the female.

*Symptoms.*—Almost any ill health in children may be popularly ascribed to worms, but definite symptoms are rare. As previously stated, the passing of the worm is usually the first evidence, although many children are suspected. Examination of the stools is rarely performed as a routine, and only a few surveys of different races and localities have been made. The presence of ova is the best diagnostic sign other than actual observation of the worm. The only gross object that is sometimes mistaken by the laity for round-worm is a long shred of intestinal mucosa. The ova may be mistaken for other microscopical bodies of similar shape.

While it is true that worms usually cause no symptoms other than the

occasional migration, vomiting or passage of one or more of them, the opinion of their potential harmlessness must be revised, with the increasing number of cases reported where they have caused appendicitis, intestinal obstruction or penetration of some vulnerable region.

The subjective and objective symptoms are indefinite, although such evidences of abnormalities should not for that reason be disregarded. The child may complain of nausea and abdominal discomfort either from colic or distention.

Disturbances which have been noted in infested children are the abnormally large appetite for food and drink. Anemia, increase in the percentage of eosinophils and the development of undernutrition are found in severe infestations. Nervous symptoms of restless sleep, fever, grinding of teeth, night-terrors and convulsions are often ascribed to this origin. When large numbers of parasites are present or migration occurs it is only reasonable to assume that they can produce symptoms, and the relief of impaired health after the removal of worms is the most convincing evidence of their influence.

Complications occur both within and remote from the intestine. Although migration is relatively rare in this rather frequent infestation, there are numerous records of invasions of accessible and what would seem to be inaccessible regions. There seems to be no cavity of the body which may not be invaded. Traveling along the alimentary tract, a worm may enter the appendix, a diverticulum, the bile or pancreatic ducts; it may ascend to the pharynx and gain entrance to the larynx, trachea, bronchus or the nares, eustachian tube and the ear. In these unusual locations, severe disturbances are produced, relieved only by the spontaneous discharge of the worm or by operative removal. Jaundice, liver abscess and peritonitis have been due to round-worms. Another serious complication is the penetration of the stomach or intestinal wall, the liver and the large veins.

In a case reported by Gallie and Brown, occurring in a child of two and one-half years, symptoms of obstruction with abdominal pain, rigidity and tenderness of the upper right quadrant and a tumor mass transversely below the umbilicus were found at operation to be due to pancreatitis caused by a worm in the pancreatic duct. The tumor was composed of a mass of omentum, peritoneum and small intestine.

There have been collected at least thirty cases of acute intestinal obstruction from round-worms. This complication follows in most instances the administration of too large doses of santonin, from the action of which masses of worms become coiled in the small intestine.

Two children, aged three and four years (reported by Watkins and Ross), received 3 and 4 grains (0.2 and 0.26 Gm.) each of santonin and calomel. In a short time vomiting occurred, the bowel became obstructed, and a sausage-shaped tumor could be felt in the abdomen. At operation the ileum was emptied of worms, following which recovery occurred.

A nine-year-old boy had a history of intestinal parasites, and developed acute abdominal distention, pain, constipation, vomiting of worms, the stools containing ova. The blood-picture was that of a leukocytosis with 88 per cent of polymorphonuclear cells. Death was due to obstruction and asthenia. At autopsy round-worms were found in the stomach, duodenum and jejunum, the latter being tightly packed with masses and coils. The intestines were filled for a distance of eight feet.

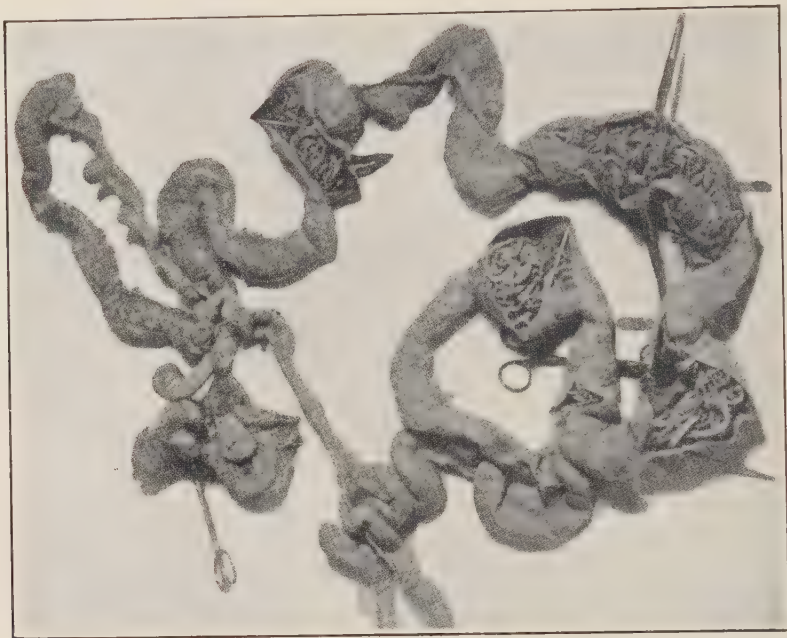


FIG. 51.—INTESTINAL OBSTRUCTION DUE TO MASSES OF *ASCARIS LUMBRICOIDES*  
(Courtesy of Sidney H. Glass, Charles J. Bloom, and *Arch. Pediat.*, 1925, 42: 589.)

*Treatment.*—In this type of infestation injections are of no use. With a preliminary light diet for two days, and finally no evening meal, the child is given 1 grain of santonin and  $\frac{1}{2}$  grain of calomel at bedtime. The following morning a dose of saline laxative or castor oil is administered. This treatment should be repeated every other night for four times. When numerous worms have been passed, the stools should be examined until no further ova are found.

The objection to santonin is in overdosage, it being wiser to give repeated small amounts of not over 1 grain. Not only may obstruction be caused by collection of large masses of worms, but xanthopsia (yellow vision), vomiting and convulsions have been reported from large doses of santonin, 3 or 4 grains at a time. I have observed the occurrence of a yellowish-red urine which persisted for several days, but with no ill effects.

The usual results of treatment are good and cure is established when faithfully carried out. Numerous other remedies have been recommended. Oil of chenopodium has been much used, the dosage being  $\frac{1}{2}$  drop for each



year of age, given once daily. The reports of toxic symptoms following its administration make it undesirable. It is better to limit one's medication to the one drug which is uniformly successful; namely, *santonin*. Intestinal obstruction accompanying the use of a vermifuge should receive prompt surgical attention. The discovery of worms in unusual situations is usually accidental at operation or autopsy.

***Necator americana* (Hookworm).**—This variety of the nematodes has been common in the southern part of the United States, South America and Africa, but the public health measures so actively employed are rapidly lowering the incidence. Infection with the parasites is not synonymous with hookworm disease, the latter being due to the severity of the infestation. If the number of worms are few, as for instance less than twenty-five, no symptoms may be present. The parasites do not multiply in the intestine, but the number is increased by repeated or multiple infections. When sufficiently large numbers accumulate, the disease manifests itself.

The larvæ enter the body through the skin of the feet, affecting the population of those regions where the children are in the habit of going barefooted. They migrate through the blood stream to the lungs. From there they are coughed up, swallowed, and reach the small intestine where they develop. The female worm, measuring 7 to 18 millimeters ( $\frac{1}{4}$  to  $\frac{3}{8}$  inch), is about twice the length of the male, much similar in gross appearance to the pin-worm.

The disease is one of the most severe caused by animal parasites. The chief symptom is the profound anemia, and the increase in eosinophils in the blood. Edema and dropsy develop. Children have poor growth and they become lethargic with poor appetites or the desire for unusual articles such as dirt, hair, cloth, etc. Younger children do not show symptoms of the disease, because of the slight infestation which has developed at this early age.

Reverse peristalsis from the duodenum has been reported in numerous cases as due to the irritation from hookworms. Henderson records the case of a ten-year-old girl with symptoms of cyclic vomiting, cured by active treatment for the hookworms.

The diagnosis is made by the discovery of the eggs or the worms. The eggs are found by diluting a portion of the stools with water, placed on a slide and observed through the microscope. They are oval in shape, surrounded by a membranous shell and contain a multiple number of cells. The worms are obtained by giving a vermifuge, either *thymol* or oil of *chenopodium*, following which they may be recovered by straining the stool through gauze. The worms are red and full of blood.

**Treatment.**—The procedure is much similar to that used in eradicating tapeworm. After a preliminary starvation, the child is given a saline laxative, and after the evacuation, *thymol* is administered in two doses, two hours apart. The dosage for children is from 5 to 10 grains in capsule or given



in a mixture to disguise its disagreeable taste. The saline laxative should again be given within one hour and treatment repeated if necessary.

The prevention of the disease is most important because of the endemic nature of the occurrence. The two methods of control have been the disinfection and care of dejecta and the discontinuance of the practice of going barefooted.

**Trichocephalus dispar (Whip-Worm, Thread-Worm, Trichuris.)—**

Although common in England and infrequently reported in this country, this animal parasite is the most common of all varieties and is world-wide in distribution. It was found as the most frequent infestation (35 per cent) of institutional children examined by DeBuys and Dwyer. The most common age is from eight to thirteen years. It is white, like the pin-worm, but is much longer, being over an inch in length, and resembles a hair. The eggs are lemon-shaped. This worm usually grows in the intestine together with the pin-worm. It is curled upon itself and has the head buried in the mucous membrane of the large intestine, usually the cecum. Its symptoms are indefinite, such as diarrhea, enteritis, vomiting, anemia and nervous manifestations, but the worm is seldom if ever seen except at autopsy.

**Tænia (Tapeworm).—**The three large varieties of the tapeworm or cestoda are the *Tænia saginata* or beefworm, the *Tænia solium* or porkworm and the *Bothriocephalus latus* or fishworm. These vary in length, in the characteristics of the segments, heads and ova. For a description of them the reader is referred to the work of Cammidge and others. These types are seldom found in children.

The variety of tapeworm peculiar to childhood is the *Tænia nana* or dwarf tapeworm. It is from  $\frac{1}{3}$  to 1 inch in length, and much resembles, in shape and segmentation, the large varieties of tapeworm. It dwells in the ileum and the number may be in the thousands.

The characteristic of all of the tænia is the intermediate or cysticercus stage of development which takes place in the flesh of lower animals, and after ingestion by man attaches itself to the wall of the intestine, where it begins to develop as a segmented parasite.

The differentiation of the different varieties is of little use, because of the similar treatment.

Tapeworm is seldom found before the age of three years. The dwarf tapeworm is reported as common in the region of New York. In New Orleans the eggs were found in 9 per cent of institutional children. The symptoms do not always suggest the cause, and the clue is obtained by the appearance of segments in the stools. Abdominal pains and nervous symptoms of restlessness and night-terrors should make the physician think of the possibility of animal parasites in the intestine. With such a history, I believe we are justified in giving a course of vermifuge treatments more frequently than we do.

*Symptoms.*—The symptoms, while not distinctive, are offensive breath, abnormally large appetite and diarrhea. Loss of weight may occur and the blood may show a high degree of anemia with an increase from the normal 1 to 4 per cent of eosinophils to many times that amount.

*Removal.*—The worm attaches itself to the mucous membrane and the head is covered by mucus and food remnants. Therefore it is necessary to clean out the intestines before treatment is begun. Magnesium sulphate or citrate is given for each of three days, with soapsuds injections each night. The child is fed only fruit juices and a small amount of milk so that there will be little fecal matter.

On the third night no supper is given and no breakfast on the following morning. Oleoresin aspidium, 10 minims, is then given in a teaspoonful each of syrup and acacia, with 5 drops of chloroform, every hour for six doses, followed by a tablespoonful of castor oil. It is customary to have the worm passed into a basin of water, and the water filtered for the examination of the specimen.

The head is only slightly larger than the neck, the latter being identified by its smooth, unsegmented, thread-like nature. The bowel should be free of all food remnants, otherwise the head of the worm may not become detached, and complete evacuation of the worm not accomplished.

At the end of three months the stools should be watched for segments, as it takes this amount of time for another worm to develop from the retained head.

**Amebic Dysentery (Infestation with *Entamoeba histolytica*).**—It is interesting to find that the first demonstration of ameba as a parasite affecting the human was from the intestinal mucus of a child. This was found at autopsy in 1859 by Lambl. Since then, this form of dysentery has been separated from the other types. The disease has been produced experimentally in dogs, cats, and rats. In this country the disease is rare in children, the usual ratio compared to adults being one under twenty-one years to ten over that age. In tropical or semitropical countries dysentery is a great devastator of child life. At Alexandria and throughout Egypt more than one-third of the gastro-intestinal troubles of childhood have recently been assigned to amebic dysentery. The manifestations in detail are not clearly or generally understood as a disease in early life. While the physician in the tropics is more especially interested, amebiasis may be imported to any country. The disease is important from the diagnostic standpoint at a time of life when inflammatory diarrheas are so prevalent. It offers a therapeutic treatment which is specific, something that is lacking in the other types.

*Etiology.*—Most observers deny the existence of amebic infection in the first year of life, and regard it as rare during the second year. The recent study of the disease in Egypt, reported during 1925, shows, how-

ever, that infants are not infrequently the victims. Petzetakis, at Alexandria, reported ten cases ranging from three to twelve months of age. He states that the infant from birth is exposed to infection and after one year of age it is one of the most common diseases. This cannot be said of France, Germany and England, but is true of the southwestern countries of Europe. Cases among children in this country are sporadic. Amberg, in 1901, reported seeing five Baltimore children affected.

The social condition is a factor, as the disease is most frequently found among the children of the poor. Dirty living conditions are usually blamed, one of the accompaniments of poor hygiene being infestation with rats, which some observers believe may be a carrier of amebæ. In regions where the disease is endemic, cases will be found, however among the well-to-do, and even in the breast- as well as bottle-fed infant.

There is a seasonal tendency for the onset in the warm months of the year, beginning in April and extending through September. This holds good in the disease as reported in Egypt, although a smaller number may begin even in the winter months. The increase in atmospheric temperature is not thought to be so much of a factor as its association with humidity. Abundance of flies is noted as a cause for the large number of cases in August and September. Epidemic influences must be regarded as factors in those regions susceptible to this disease. As the disease is apt to become chronic and to last through several years, the time when the individual became infected may be difficult to determine.

The causative parasite, the *Entamæba histolytica*, is carried by drinking water from surface drainage, especially into wells. It has been successfully grown in an artificial medium which contains bacteria and red blood-cells. A suitable artificial medium for growth is an alkaline preparation known as the Locke egg-serum, at a temperature of 27° C. (80.6° F.), which even after much time does not destroy the virulence of the organism. The identification of the ameba from the stools is difficult if the organism is dead, as it resembles large round epithelial cells. While alive the parasites assume numerous shapes, and are characterized by their movements and ability to migrate. They have been recovered from lesions in the colon, in abscesses of the liver, lungs and lower jaw. The amebæ live usually from two to six hours after passage.

*Lesions.*—The intestinal changes are nearly always confined to the large bowel, and consist of a small ulcer in the mucous membrane with considerable undermining. As the disease progresses, the wall of the intestine undergoes thickening; in some areas marked thinning. Abscesses adjacent to the intestine are found at autopsy. Perforation, diffuse peritonitis, adhesions and deformities of the intestine are frequently reported. The layers of the intestine may become dissected, honeycombed, and shed as sloughs which appear in the stools. The stools contain much mucus, blood and undigested food.

*Symptoms.*—The manifestations depend upon the acuteness or chronicity, as well as the severity of the disease.

In young children, the acute gastro-intestinal form is the one usually seen. In the mild case, the symptoms are more apt to be preceded by two or three days of anorexia, some vague digestive discomfort and diarrhea. The disease may, however, be sudden in onset and be suggestive of pneumonia. Fever is moderate, from 100° to 102° F. (38° to 39° C.). The pulse and respiration are increased, the extremities cold, and occasionally acetone will be present in the urine. The child vomits or has hiccoughs, the stools become frequent, mucopurulent or glairy with mucus. Blood may appear as streaks or in considerable amount. The number of stools is variable, from ten to forty in twenty-four hours, sometimes occurring every ten minutes.

An anginous form is found in children, beginning with redness and swelling of the pharynx and tonsils, and at first an absence of intestinal symptoms.

In some cases diarrhea is pronounced, with peculiarly green watery stools. All the general symptoms are exaggerated. After a few days the character of the infection may be suspected and the parasites found.

Subacute cases are recognized with difficulty. After several days of indefinite gastro-intestinal symptoms, such as loss of appetite, but with little or no fever, the stools show a moderate increase in number, and in mucus and blood.

Severe acute forms resemble meningitis or typhoid fever. The general condition is one of septicemia, and the local one of hemorrhage and gangrene in the intestine. The disease begins after an indisposition of forty-eight hours, with high fever, bloody purulent discharges. Soon the child passes into a state of coma, the pulse is weak, irregular, the extremities cold, and convulsions appear. Lumbar puncture is negative except for increased pressure. The stools may be prune-colored or hemorrhagic, putrid and gangrenous in odor. Death results in a few days.

A chronic form resembles chronic ulcerative colitis or chronic diarrhea. It begins usually after the age of two years. Chronic cases are the despair of the physician because of the tenacity. In some instances there is a history of previous dysentery in the parents. At times the stools may be constipated, or they may alternate with diarrhea. The discharges vary greatly and may be pasty, undigested or serous, fermented, liquid, mucous or streaked with blood. The examination of the stools for ameba is difficult and must be repeated if unsuccessful. The previous administration of calomel is advisable before looking for the parasites in the movements. Sometimes the abdominal pain is suggestive of appendicitis, and indeed the appendix has been found invaded.

The blood shows an increase of the white cells, chiefly polymorpho-



nuclears. The hemoglobin is somewhat reduced, the red count little affected.

As a result of the chronic infection, the child's condition is a serious one, characterized by definite anemia, cachexia and the occurrence of relapses. The younger the child the more serious the outcome. In infancy the disease is almost uniformly fatal.

In older children there may at first be no discomfort, and the child be happy and little affected. In the well-developed case, there is abdominal pain, distention, enlarged liver, straining at stool, with four or five discharges daily, containing blood or clots. Prolapse of the rectum may occur. General weakness results from insufficient food and from the exhausting diarrhea. The temperature is little disturbed. Edema of the extremities is present in some cases. The general condition is what would be expected in a chronic and relapsing diarrheal disease. Abscess of the liver is not so common an occurrence in children. The death-rate in early childhood is high. Complications are rare, but amebic bronchopneumonia has been reported. Tenesmus of the bladder and cystitis may occur. Transient pain in the long bones is complained of. Ulcerative stomatitis appears as an unfavorable symptom. Amebic encephalitis or meningitis is indicated by apathy and photophobia, and is an evidence of severe and fatal outcome. Acute myocarditis is not uncommon, with a cardiac murmur.

Convalescence is rapid in some of the treated cases, but usually the older children are months or years in getting well. The acute cases with relapses pass into the chronic form. There is a predisposition to other infections.

*Treatment.*—Castor oil is to be given at the onset. When the stools are putrid, and there is colic or tenesmus, a high injection of flaxseed meal decoction gives relief. Irrigation of soluble quinin in strength of 1 : 5,000 or more has been in much favor. The opinion as to its efficacy is now not favorable.

Ipecac by the mouth was formerly the specific treatment. This has now been largely replaced by emetin upon which is placed the chief reliance. Emetin sometimes causes toxic manifestations due to its cumulative effect, the symptoms of which are tachycardia, loss of appetite and, less commonly, paralysis. Therefore the repeated doses should be carefully watched. A course of treatment comprises six daily injections, subcutaneously in infants, and intramuscularly in older children. Under one year the dose ranges from  $\frac{1}{12}$  to  $\frac{1}{6}$  grain (.005 to .01 Gm.), from one to two years  $\frac{1}{6}$  to  $\frac{1}{4}$  grain (.01 to .015 Gm.), from three to ten years  $\frac{1}{3}$  to  $\frac{1}{2}$  grain (.02 to .03 Gm.), and from ten to fifteen years  $\frac{1}{2}$  grain (.03 Gm.).

A blood transfusion is of great benefit at the onset of the treatment. From 8 to 16 ounces should be given, according to the size of the child.

As a result of this treatment the stools become formed, free of blood and ameba. Because of the probability of relapse another course of treatment should be given at the end of two months.

Atropin is indicated for the tenesmus, hot baths for hygiene, and cool enemata for comfort.

The diet should be nutritious, with buttermilk, albumin milk, much sugar and other nourishing foods. Cod-liver oil and iron will be of help in combating the anemia and in restoring body weight.

**Infestation with *Lamblia* (*Giardia*) *intestinalis*.**—This animal parasite is motile, pear-shaped, flagellated, of microscopic size and is sometimes reported in the stools of children. It lives in the small intestine attached to the epithelium.

The parasite is seen during diarrheal disease, possibly as a secondary invader. It is carried by the water from small animals such as mice, rabbits, cats, etc. There has been much doubt thrown upon its pathogenic nature, but its presence in three children seen recently, who for months had indefinite looseness of the stools but considerable blood therein, make me believe that a mild dysenteric-like disease can be caused by infestation with *Lamblia*.

The treatment is similar to that of amebic infection.

**Infestation with *Balantidium coli*.**—This ciliated parasite is also of microscopic (0.1 Mm.) size, oval or round in shape, carried in swine manure. It is found chiefly in the colon, in great numbers, and may cause catarrhal inflammation of the intestines. It is a rare cause of dysentery, resembling the amebic type, and lasts for years.

The parasite lodges in the tissues but it can travel rapidly, due to the cilia which cover its surface and to the transverse muscular bands.

The symptoms are undigested, mucous, bloody stools, with straining and griping. DeBuys reports a child cured by emetin which he regards as a specific.

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## CHAPTER XXV

### POISONING

#### ACCIDENTAL POISONING

Accidental poisoning is prevented in the early years of childhood only by the utmost care in keeping poisons out of the reach of the restless, investigating and meddlesome child. The practical method of doing this is the exclusion of poisons from the household. In actual practice there is often some question as to the character and amount of the poison swallowed. The panic which the family experiences and the difficulty in finding and administering the proper antidote cause a delay which may make recovery impossible. It is not probable that a child with a caustic solution in one glass and the proper antidote in another could swallow both of these consecutively without a severe burn resulting. It is hopeless, therefore, to expect escape from severe damage by the use of an antidote which is unsuitable or delayed for minutes or hours in administration. Theoretically it would seem advisable to require druggists to dispense the antidote in an accompanying container when a poison is sold to the laity. Enforcement of this would be difficult, and the administration of the antidote would often be found impossible by the untrained layman. Prohibition of the sale of all dangerous poisons to families is the only safeguard that children can secure, as the family may be careless as custodians of these articles.

Many practicing physicians carry in their satchels a small handbook of advice for the administration of antidotes. Such will be found of the greatest use in emergencies, but a small case of antidotes ordinarily needed is a more practical method. Under the subject of toxic gastritis are mentioned the general household measures which are recommended for immediate treatment of ingested poisons. More detailed directions as to the administration of antidotes are the following:

**Alkalies.**—Caustic alkalies of sodium, potassium and ammonia are present in concentrated lye and washing powders. Their action in the body is corrosive, causing pain, inflammation, spasm of the pharynx and esophagus, with destruction of tissue. Ulcerations and strictures persist in the esophagus and stomach in those cases not at once fatal. Treatment should consist of prompt neutralization of the alkali with large amounts of lemon juice or vinegar. In addition, butter or oil should be given with much milk or water to dilute the poison. In case strong solutions of lye have been swallowed, the stomach tube should be used only with great care,

because of the danger of perforation of the esophagus or stomach. The pain is to be relieved by morphin hypodermically. Collapse is combated by stimulants. The cicatricial deformities cause embarrassment to feeding, and result in much suffering and loss of weight, requiring long-continued special treatment.

**Acids.**—Sulphuric, nitric and hydrochloric acids produce erosion and marked tissue destruction in the digestive tract and death occurs from perforation, shock or suffocation within a few hours or days. The treatment is by prompt neutralization and dilution of the acids with soapsuds or magnesium preparations, both of which are usually available in the household. These should be given in large amounts of milk, water and eggs. The laryngeal swelling produces marked interference with respiration and in such complications tracheotomy may be necessary.

**Carbolic Acid.**—Phenol in watery solutions is not markedly acid; it may be neutral. Proprietary mixtures of carbolic are kept in most households for disinfecting or antiseptic purposes. The swallowing causes pain in the throat and stomach. Burns are of the nature of a necrosis, as the action is one of protoplasmic poison rather than a corrosive irritant. Pallor, collapse, cyanosis, convulsions and nephritis are manifestations which appear promptly in the serious cases. Death from failure of the respiration results within twenty-four hours. Treatment should be by lavage with 10 per cent grain alcohol, followed by large amounts of egg and water. In case alcohol is not available, a quart or more of weak potassium permanganate solution (1:10,000) is an excellent antidote. The stomach should always be emptied of all solutions and repeatedly washed out with warm water. The body heat, which has become subnormal, should be raised by application of hot-water bottles. Warm salt solution under the skin or in the vein will stimulate the circulation.

**Alcohol.**—Denatured or wood alcohol is now a household article. An ounce or less may cause death to a child. Severe abdominal pain soon follows its ingestion and the other toxic manifestations are nausea, vomiting, inflammation of the stomach and small intestine, circulatory and respiratory failure and death in from one hour to two days. Not all cases die, but blindness is apt to appear before death, or to develop in those who live.

Prompt and repeated washing of the stomach with soda bicarbonate solution is indicated. Much water is to be administered. Strychnin should be given in regular doses and a purgative to increase elimination.

**Arsenic.**—Acute poisoning by arsenic occurs from swallowing Paris green, rat poison and by overdoses of Fowler's solution or arsenious acid. Toxic manifestations from arsenphenamin administration are usually of a chronic nature. It makes little difference what preparation is used or how it is given; the actual poisoning is caused by the liberation of arsenious acid in the body. Taken by the mouth arsenic causes inflammatory symptoms in the gastro-intestinal tract. The child will soon have difficult swallowing,



nausea, bloody vomiting, pain in the stomach, diarrhea, water loss, diminished urine, nervous symptoms and collapse. Fatty changes occur in the liver and kidney. Death results from exhaustion or starvation.

*Treatment.*—Copious lavage with warm water should be repeated to free the stomach of the arsenic. Epsom salts should then be given. The body should be kept warm and stimulants such as coffee or digitalis administered. Much water by mouth or under the skin is advisable.

For the specific treatment of arsenical dermatitis, sodium thiosulphate has been extensively tried by McBride and Dennie (see mercury poisoning).

**Aspidium and Oil of Chenopodium.**—These vermifuges in overdoses may cause gastro-intestinal irritation. Lavage, saline laxatives, milk and albumin water are indicated if toxic symptoms appear. Convulsions should be treated by chloral or bromid and in case of collapse, strong coffee.

**Atropin.**—Fatal belladonna poisoning is rare and the action of the drug is slow as compared with other poisons. Death is by asphyxia within twenty-four hours or longer. I have known of numerous children receiving atropin in large amounts where teaspoonfuls of solutions were given instead of drop doses, and where the child gets hold of the bottle containing aloin, strychnin and belladonna. Thus far I have known of but one death. Among the symptoms of poisoning are dryness of the throat, difficult swallowing, nausea and vomiting. Scarlatiniform rash is a constant diagnostic manifestation. The treatment is usually successful, consisting of emesis, lavage with weak tea and the hypodermic injection of  $\frac{1}{20}$  grain of pilocarpin in small children and  $\frac{1}{40}$  in large-sized children. Strong coffee, artificial respiration and ice applied to the head are of value.

**Camphor.**—Occasionally camphor will be swallowed accidentally by the child in some form commonly kept in the household. Camphorated oil may be ignorantly or inadvertently administered by the mouth, and has caused death within seven hours. Camphor causes stimulation and later paralysis of the brain centers. Convulsions may develop within a few minutes after its ingestion, and consciousness not regained. The local symptoms are a burning sensation in the pharynx, esophagus and stomach, the development of thirst, nausea and vomiting. The urine becomes suppressed and the child dies from asphyxia and collapse within twenty-four hours.

Pathological changes are not distinctive for camphor poisoning. The esophagus is not seriously injured, nor is there marked evidence of irritation anywhere. Small hemorrhages are found in the stomach, intestines and kidney.

The treatment is the prompt administration of any household emetic and castor oil, and the induction of emesis by the introduction of the finger into the pharynx. The physician should promptly introduce a stomach tube, some olive oil and then siphon out the contents. Bromid and chloral are used for the convulsions, and to prevent collapse, coffee or other stimulation by the mouth and external applications of heat should be administered.

**Formaldehyd.**—This disinfectant is commonly kept in the home, but is rarely swallowed by children. It causes ulcerative gastritis when ingested in concentrated solutions. The local symptoms are inflammation of the mouth, abdominal pain and diarrhea. Acute disturbances of the central nervous system, suppression of the urine and hematuria commonly result. Rapid recovery may occur, or death within one or two days.

*Treatment.*—Prompt lavage of the stomach with dilute ammonia water is to be performed.

**Lead.**—Children may eat fresh paint or from habit chew the dried paint on toys. Lead and opium lotion is kept in some households. Intoxication may be caused from drinking water from lead pipes. Wrappers from foods may contain lead and contaminate the contents. Lead acts as a gastro-intestinal irritant. Poisoning produces blue gums, thirst, severe nausea and vomiting, abdominal pain, gastro-intestinal paralysis, constipation or diarrhea and black stools. The other symptoms are palsies, fatty degeneration of the muscles, anemia, coma, convulsions, suppression of urine. Death may occur in twenty-four to forty-eight hours from inflammation of the stomach, intestines and kidneys.

*Treatment.*—The stomach should be emptied by vomiting or lavage, and Epsom salts given. Atropin and opium are needed for the colic.

**Mercury.**—Bichlorid of mercury tablets or solution are found in many homes, and may be swallowed by the child. The intoxication from mercury ingestion is produced by absorption from the mucous membrane of the gastro-intestinal tract. This is inflamed and becomes corroded, and nephritis is produced. The mouth shows evidence of the poisoning by salivation, grayness of the mucosa, pain and swelling. There is marked thirst. The intestinal symptoms resemble those of lead poisoning. Death may occur in an hour or not for weeks.

The treatment is by prompt administration of white of egg or milk. This should be vomited or washed out of the stomach. Lavage and intestinal irrigation should be practiced daily. Milk and cream of tartar are to be given every two hours. Hot packs for sweating are indicated daily.

Sodium thiosulphate intravenously and by the mouth simultaneously is recommended as an antidote for bichlorid in acute poisoning. Sterile ampules ready for use may be obtained. The initial dose for a six-year-old child is 0.15 gram (1 grain), on the second day 0.22 gram, on the third day 0.3 gram. A pint of water is given by the mouth in twenty-four hours, and to this may be added the thiosulphate.

**Opium.**—Fortunately there is now little menace to childhood from preparations of opium, since these are not obtainable, with the exception of paregoric, which is not of great danger in the small amount to which the child is exposed. A fatal outcome is due to respiratory failure. No gastro-intestinal symptoms other than vomiting, nor any characteristic pathological lesions are found.

*Treatment.*—Prompt and repeated lavage with 10 grains permanganate of potash per quart of water. Artificial respiration and stimulation by hypodermic of caffein or strychnin are needed for stimulation for one or two days. Laxatives should be given until the bowels have moved well.

**Santonin.**—This drug is taken in prescriptions. Seldom does the physician give a dose which causes intoxication, but it is taken in over amounts by ignorance of the proper dosage. Deaths from respiratory failure in children, within one to twenty-four hours, have resulted from 2 to 3 grains at a dose. Poisoning causes gastro-enteritis, a peculiar yellowish or purplish urine, disturbance of color vision and of the other special senses, convulsions and collapse.

*Treatment.*—Gastric lavage, to be repeated if symptoms have occurred within an hour or two following ingestion of the santonin. Epsom salts may be given twice daily. Convulsions or collapse require sedatives or stimulation.

**Phosphorus.**—Formerly children were poisoned by chewing phosphorus matches. These are no longer sold. Rat poison is kept in many homes. Certain fireworks, notably "spit devils," contain phosphorus. These have promptly caused death whenever eaten. Death occurs in acute severe poisoning within twenty-four hours. The visceral changes are catarrhal gastritis and subendothelial hemorrhage in the heart and blood-vessels, fatty degeneration of liver, kidneys and heart. If the child lives long enough, the stomach undergoes necrosis, perforation and gangrene.

Experiments made by Dwyer and Helwig upon dogs which were given a lethal amount of phosphorus showed that the animals could be saved within two hours by administration of large quantities of mineral oil.

Children should be given 3 ounces of mineral oil. The stomach should be promptly emptied by lavage with 1 : 1000 permanganate solution which forms phosphoric acid. In case there is at hand peroxid of hydrogen, it may be used, 1 ounce to 2 quarts of water. More oil should be given after lavage. *Do not use castor or other vegetable oil.*

**Silver Nitrate.**—Lunar caustic and silver solutions are obtainable by children in many homes. Applications of the stick to oral lesions are dangerous in children, as pieces may break off and be swallowed. The caustic effect is shown by marked pain in the pharynx, esophagus and stomach and by vomiting, inflammation of the stomach or intestines, resulting in blood-streaked, diarrheal stools. The small amount of silver nitrate usually swallowed is insufficient to cause death, but in large quantities convulsions may occur, followed by coma.

*Treatment.*—The silver nitrate swallowed should be at once neutralized by salt and the stomach emptied and irrigated with salt solution. Milk with beaten egg is then to be given.

**Turpentine.**—The swallowing of turpentine causes symptoms of gastrointestinal inflammation. It slows the respiration and causes mental excite-



ment. Urination becomes painful, scanty, the urine containing blood and albumin. Death within a day has occurred in children from quantities less than an ounce.

The turpentine should be removed by repeated gastric washing, and for several days olive oil or mucilage of acacia given. Stimulants such as coffee should be administered.

## FOOD POISONING

Toxic substances of various kinds when present in the food cause manifestations of poisoning. Elimination of drugs through human milk may occur when given in large or long-continued doses to the mother, and be responsible for rashes and digestive discomfort in the infant. Some of the drugs which have been found to affect the child in this way are bromin, belladonna, iodine, salvarsan and salicylic acid. No treatment is needed other than withdrawal of the offending substance from the mother's diet.

Acute gastro-intestinal poisoning not infrequently occurs from contamination of the food with certain microorganisms of the colon bacillus type. According to the present opinion, the causative agent is not a ptomain. The most common microorganisms found in such cases are the paratyphoid and the botulism bacillus. It is probable that epidemics of vomiting and diarrhea in the winter months are, more often than we suspect, due to infection with paratyphoid organisms. *B. proteus* also produces gastro-enteritis with hemorrhage, high fever, delirium, muscle and joint pain and transient paralysis of the extremities.

**Botulism.**—The frequent occurrence of botulinus poisoning has recently received much attention and is significant because of the great increase in the use of preserved foods. Botulism is a fairly common poisoning, 147 outbreaks having been noted in America, due to improperly prepared food which has been insufficiently heated to destroy the anaërobic microorganisms or their toxins. *B. botulinus* is an inhabitant of the garden soil. Over one hundred strains of types A and B have been isolated from human and animal sources. These come from soil, plants and canned-food products. Vegetables canned at home by the cold pack method are especially liable to be dangerous for use. String beans, corn, asparagus, peas, pickles, spinach, figs, olives and some meats such as sausage, meat pastes, sardines have been responsible for epidemics or isolated cases. Thorough cooking will destroy the bacillus as well as the toxins.

The seriousness of the poisoning is due to the development of lesions particularly in the brain stem, producing paralysis of the bulbar type. This resembles the medullary paralysis of poliomyelitis, though the minute pathology is not identical. Fatty degeneration of the liver and hyperemia with paralysis of the intestines occur. Macleod regards the action of poisonous products developing in the food as somewhat similar to the more



chronic intoxication from protein decomposition of substances in the alimentary tract, such as are found in intestinal stasis and obstruction.

*Symptoms.*—In from one to five days after eating food contaminated with the botulism bacillus, gastro-intestinal symptoms appear. Although these are not the most serious manifestations of the disease, they are quite definite. They consist of nausea, vomiting, abdominal pain, scaphoid abdomen, diarrhea or constipation. The breath gives off the odor of spoiled cheese. The characteristic disturbance is the difficult swallowing, due to constriction and paralysis of the throat muscles. Later, paralysis of the intestinal tract occurs. Accompanying the dysphagia is the difficulty in speaking and the development of dyspnea and asphyxia.

The early stage is characterized by intoxication, fatigue, headache, dizziness and weakness resembling paralysis. Marked ocular disturbances are the impairment of vision and the dilatation of the pupils. Stupor, or insomnia and restlessness may be present. There are disturbances in the memory, hearing, and cutaneous sensations, and there may be pain in the neck. These manifestations are almost identical with the bulbar type of infantile paralysis.

The skin becomes dry, the urine decreased. The temperature remains below normal. The heart is weakened and may fail in company with the respiration. The disease lasts from one to three days, occasionally six, and death occurs in two-thirds of the cases.

*Treatment.*—Prophylaxis consists in the thorough boiling of all vegetables and meats at the time of canning. The safest method is to thoroughly cook all canned and dried foods at the time of using. Preserved foods which are spoiled do not always give evidence by their appearance, odor or taste, but it is wise to discard all preparations in which abnormalities are evident.

Active treatment should be promptly begun, by emesis and gastric lavage. Epsom salts or castor oil should be given to cause early elimination. Thorough purgation is necessary when obstipation is present, because of the poisonous content of the ingested food. Saline enemata are indicated. Rest in bed as practiced for infantile paralysis is necessary. Because of the difficulty in swallowing, fluids should be given under the skin. As soon as food can be taken, liquids should be fed.

Strychnin and pilocarpin are useful for the stimulation of the circulation and the secretions. The subnormal temperature calls for applications of external heat. Dyspnea is to be treated by inhalation of oxygen.

A few communities are supplied with botulinus antitoxic serum. Such has been prepared from animals which have been injected with non-fatal doses of the specific virus. When it is possible to secure this antitoxin its early use is advisable.

**Alimentary Allergy.**—Children are especially sensitive to reactions from foods commonly given at their time of life. The clinical manifesta-

tions may be confined to the gastro-intestinal tract, or be accompanied by local cutaneous lesions, such as urticaria, erythema, angioneurotic edema or eczema; by sudden attacks of allergic asthma; by constitutional symptoms of fever, pallor and shock. The idiosyncrasy to cow's milk is now recognized as allergic.

The discovery of the phenomenon of protein allergy has been followed by the recognition of numerous provocative substances and clinical manifestations. Some of the alimentary reactions are striking and can be accepted as specific, in the case of egg and milk especially.

In 1831, pollen was first recognized as the cause of hay-fever, and only in 1884 was it found that digestive disturbances, such as nausea, vomiting and abdominal pain could result from idiosyncrasy to food. It is remarkable that the allergic cause of angioneurotic edema remained unrecognized until the last few years.

*Susceptibility.*—Several observers have reported that about 10 per cent of all children carefully tested by the cutaneous method give positive local reactions to one or more proteins. Only a small percentage of the reactors have clinical symptoms, and some of these may be caused by substances other than the particular ones used in the test. Food sensitization usually begins in the early years of childhood and tends to disappear spontaneously in the later ones.

The influence of heredity upon the causation of sensitization is as yet little known. From the figures of the writers who discuss the subject, it seems that from 15 to 20 per cent of asthmatic individuals have a positive history in one or both parents. There is a higher incidence in hay-fever (allergic rhinitis). That there is an influence of this nature upon the production of intestinal allergy is possible. While not present in all cases, it has been definite in some families I have known. A neuropathic diathesis may play some part in the causation. A difference in susceptibility was found in a pair of twins recently seen, one of whom had eczema; the other never had it. Both were being nursed exclusively from the same mother. It is hard to explain such a variance.

The sensitizing agent may enter the body by contact, inhalation or the feeding, usually the latter. Sensitization by the inhalation of emanations from pollens, rabbit hair, house dust, and many others, has proven to be the most common excitant of allergic asthma. The coexistence of asthma and eczema in infants is best explained by multiple sensitization to inhalants and food. Not all asthma is allergic. One cannot explain all cases of eczema (the exudative diathesis) upon the basis of allergy. The great majority of eczematous infants show their first and sometimes their only symptoms while on the breast. Urticaria and angioneurotic edema are never caused by breast milk. That foreign protein or its derivatives can pass through human milk is beyond proof, but it can be assumed when by

exclusion of certain foods from the nursing mother, the allergic symptoms disappear.

It is generally accepted from the work of Schloss and others, that undigested protein can be absorbed by the alimentary tract of children who have gastro-intestinal or nutritional disturbances. Undigested protein, when absorbed, acts as a substance foreign to the blood. The child becomes sensitized to a specific unsplit protein, the most conclusive evidence of its absorption by the intestine being the appearance of urticaria or scarlatiniform rash during or soon after an attack of diarrhea. Eczema, often an accompaniment of nutritional disturbances, may be produced in the same way. It seems most probable then that during digestive upsets, incompletely digested protein may be absorbed from an intestinal wall which has become more pervious as a result of injury, and that the sensitized child reacts by abdominal or general toxic manifestations whenever this type of protein is fed.

The manifestations of alimentary allergy much resemble clinical intestinal intoxication. The allergic reaction is, in fact, an intoxication produced by the protein which on its repeated introduction into the body becomes an intoxicating agent.

Until a few years ago, the profession commonly prescribed albumin water, made with egg-white, as a temporary food in acute intestinal disturbances. The danger from such raw protein is now recognized. Coudat reports a catastrophe from this cause in a five-months-old infant who suffered from eczema and diarrhea. Administration of egg water caused within two hours a severe shock. Egg furnishes the greatest number of cases of alimentary allergy. An instance of the relationship of eczema and asthma to sensitization is that of an infant in my practice who at six weeks of age began to have these disturbances while on the breast alone. When sixteen months old, it was found that whenever he was fed raw egg, he immediately vomited. Other evidences of idiosyncrasy appeared, especially the swelling of the face, and a transient erythema. At two years of age he still had allergic manifestations whenever egg touched his mouth.

While proven idiosyncrasy to milk has been infrequent in my experience, there are two infants who repeatedly became alarmingly ill immediately following the drinking of a few swallows of cow's milk. The manifestations were swelling of the lips and face, vomiting, collapse, diarrhea and urticaria. Recovery from an attack took place within twenty-four hours.

Multiple sensitization occurred in a girl whose course I have followed from the age of eight to thirty years. While a young child she suffered most severe and terrifying disturbances when cow's milk or egg was taken in any form. The sensitization reached such a degree that a trace of either of these proteins in the diet caused profound symptoms, rapid swelling of the lips, tongue and throat, with difficult breathing and scarlatinal-like erythema of the skin. A spoon which had been used for serving egg would



bring on a severe attack. When I first became acquainted with this child's symptoms, I felt that suggestion or hysteria must play the leading rôle in the disturbance. However, when the suspected foods were given without her knowledge of their nature, the alarming manifestations recurred; as a result of which no further attempt to feed them was made for several years. By the age of twenty years, desensitization had occurred, and since then she has showed no further trouble and is not disturbed by any food. Her three children have never had any food idiosyncrasies.

*Symptoms and Diagnosis.*—Vomiting may begin within a few minutes after taking the particular food, and may be of a severe nature, accompanied by collapse or shock. The stools become watery, mucous or bloody, and the intestines bloated. Fever may be present. The duration of the acute disturbance is short, seldom over twenty-four hours. Recurrences take place whenever the food even in minute quantities is given. A symptom emphasized by Duke, to which insufficient attention has probably been paid, is the occurrence of local or general pain in the abdomen. Severe colic and griping may appear, or the manifestation may be indefinite and in the nature of discomfort or distress. It becomes chronic when the offending food is not restricted from the diet.

Vomiting and diarrhea are common to many other disturbances, but the history of similar attacks following the eating of a particular food permits one to recognize the specific nature of this disturbance. This does not, however, preclude the necessity for a thorough physical examination and the consideration of organic lesions such as intestinal obstruction as the possible cause of the sharp attack.

The accessory symptoms of cutaneous rashes, asthma and angioneurotic edema are not necessarily present, but add greatly to the specificity of the diagnosis.

The development of skin tests with the powdered proteins has been extensive and these tests widely used in the past ten years. The number of substances to which an individual may become sensitized is so great that one cannot expect to test for all of them; indeed, it is impracticable to submit a child to more than a few. The technic and interpretation of skin testing cannot be mentioned at this time. Reliable preparations, proper application, correct reading of results and the limitation of the tests to the ones that are indicated in the particular case are factors that must be considered. Scratch tests are without danger, but all methods are time-consuming and annoying, and the results not always convincing. The size of the skin reaction indicates an increased concentration of the test proteins rather than an evidence of the importance of the particular substance as the cause of the symptoms.

The determination of the offending food element in the infant's dietary is not so difficult until general feeding is begun, when more than one article may be causative. Multiple sensitization from feeding or inhalation is not infrequent.



The practical diagnosis rests upon the history of gastro-intestinal disturbances recurring when a certain food is used, and the freedom from attacks when the offending protein is kept out of the child's diet.

*Treatment.*—Permanent elimination of the disturbing food from the diet may be required. In some cases all that is necessary is the reduction of the amount fed. One child of my acquaintance illustrates this fact, in that she could drink a glass of milk with her meals, but attempts to take additional amounts caused her to break out with hives. One may become sensitized to the substitute protein if too large a quantity is given.

Desensitization occurs as the child grows older, often by the time the infant has reached the age for general feeding. It may be hastened in specific instances by at first excluding all of the offending protein and then by gradually feeding small amounts with an increase as rapidly as possible. Nutrition cannot long be maintained in the infant without milk in some form. Breast milk, dried or evaporated milk preparations, buttermilk, boiled milk, goat's milk, may all be tried before relief is obtained. All fresh cow's milk should be well cooked as the protein becomes less toxic by boiling, or preserving in dried form.

Subcutaneous injection for desensitization is impracticable in most children. Meat, boiled milk and cereals may be diluted and given in small amounts by the mouth. As egg often causes swelling of the mouth by contact, it is advisable in older children to give it in capsules as suggested by Blackfan.

**Milk-Sickness.**—A peculiar form of severe intoxication results from the drinking of milk from cows that have grazed upon the white snakeroot plant. It was once common in the middle and southern portions of the United States before the soil had been cultivated, and is still seen occasionally in the mountains of North Carolina. The characteristic symptom is acidosis which accompanies or follows the loss of appetite, nausea, persistent vomiting, severe constipation and muscular weakness.

The present generation of physicians little realizes what an important disease milk-sickness was in the pioneer days of the United States. In the first half of the last century whole communities were decimated, and a considerable portion of the practice of the pioneer physician was the treatment of this affection. It was found that children, nursing babies excepted, were more frequently affected than adults.

Probably no other disease was so frequently reported in the current literature during that period. In the one-hundred-page monograph by Jordan, Harris and Luckhardt from the Chicago University, in 1909, a complete bibliography is given, consisting of seven pages. Among the contributions will be found several from Germany and England in which milk-sickness was regarded as an American disease. Kimmel of Berlin so described it in 1891. "Sick Stomach of Tennessee" was the subject of an article in the *Cincinnati Western Journal of Medical and Physical Sciences*,

in 1830. One of the early writers was Drake, who reported the disease in North Carolina before 1776. The states that suffered principally, or in which reports were more frequent, were Ohio, Indiana, Illinois, North Carolina, Kentucky, Tennessee and Georgia. Jordan and Harris studied the disease in New Mexico as the basis of their communication. The disease has largely disappeared with the clearing of the land and the restriction of grazing in wild timbered sections where poisonous plants grow. The writer is indebted to the recent report of Wilkinson for calling attention to the fact that the disease still occurs in North Carolina and may be encountered at any time in certain regions of the United States. In his report there is an account of six cases, three of whom were in children from three to thirteen years of age.

*Etiology.*—The disease in man is caused by using the milk, butter, cheese or flesh of cows that have eaten white snakeroot, a poisonous plant which still exists in the woods of uncultivated regions. Many theories have been suggested as to other causes for the disease, among them poison ivy, bacterial infection, etc., but it seems proven by all experience that the cause is snakeroot. Cases which developed at a distance from the region where the animals were poisoned were known to have eaten butter, cheese or dried beef transported from there.

Cases are most numerous in August, September and October, especially in dry seasons, when the plant grows luxuriantly. The following paragraph descriptive of the plant is quoted from the article by Clay:

The plant, white snakeroot, is found in the rich soil of a thickly-shaded oak ridge near a small stream with a sandy bottom. It blooms in September, with a white blossom of the corymb type, a cluster of flowers each on its own foot-stalk and arising from a common axis. The stalk is from one to four feet high, with a square stem. . . . The leaves are petioled and opposite, broadly oval at the base, taper-pointed, coarsely-toothed, three-ribbed and veined. . . . They grow in pairs from alternate sides of the stalk, each pair at right angles to the pair above or below.

*The Disease in Animals.*—Cattle especially are affected by the disease and in the earlier days of grazing upon wild land, whole herds were wiped out. The disease in the animal is known as "trembles" because of the marked tremors which develop. Other domestic animals may be poisoned by the plant. Horses, hogs, dogs are less frequently affected than cattle. Rabbits and buzzards have also been found with the disease. The convincing proof of the pathogenesis is the production experimentally in feeding the plant to animals. This was done by Jordan and Harris who believe that the alkaloid or active principle is transmitted to humans, most commonly by milk, but also carried in butter, cheese, beef and pork. In 1918, the North Carolina Agricultural Experimental Station, through Wolf and coworkers, reported that the poisonous property is probably a glucosid. In animals the disease manifests itself in a period from a few hours to

several days. It causes paralysis of the extensor muscles of the legs, paralysis of the bowels, marked tremors, coma and death. The disease in animals is much similar to that in man.

Numerous postmortem studies have been made on the animal, but few on the human. Little is known of the anatomical changes. There probably is nothing characteristic for this disease. At autopsy there is found capillary injection of the mucous membrane of the intestine with bloody mucus lining the surface. Other findings have been fatty degeneration of the liver and cloudy swelling of the kidneys. There is no indication of involvement of the central nervous system.

*Symptoms.*—The intoxication may be acute or slow in its course. When appearing suddenly, there is loss of appetite, the stomach is persistently intolerant of all food, constipation is as complete as in intestinal obstruction, and there soon follows weakness in the extremities and atony of the muscles. Weakness, exhaustion and emaciation are progressive. The lips are red, the tongue is tremorous and inflamed, the breathing is deep and of the air-hunger type, the breath is fetid and has the odor of chloroform or acetone. The urine becomes dark, scanty and contains albumin and acetone bodies. Acidosis is therefore a prominent and constant diagnostic manifestation as shown by the syndrome just mentioned.

Hiccough and difficult swallowing are found in some cases. There may be marked thirst, but the individual cannot retain what he swallows. The abdomen which is at first retracted becomes distended. The blood shows a reduction in the number of white cells with a relative increase in the polymorphonuclears. The blood-pressure is lowered. The reflexes are diminished; convulsions and later stupor or coma may occur.

The period of intoxication is from three to five days. Tremor in the legs may appear, but is not as characteristic as the “trembles” of cattle.

Individuals who get well often require months and years for complete recovery from the weakness. Death from the disease occurs in 10 per cent of the cases.

The chronic form presents the same type of symptoms, but they are less active. Overfeeding or exertion brings on acute manifestations, so that relapses occur. Recovery may take years, and some individuals seem to be permanently invalidated by the disease.

*Treatment.*—It is natural that the treatment of the disease in a rural and inaccessible district should have been by remedies which are easily obtained. These are whiskey and molasses. Alcohol or wine has been used empirically to the point of intoxication. Clay believes that alcohol has an affinity and a neutralizing effect upon the poison. Glucose in the form of molasses or honey is given in large quantities from 4 to 6 ounces every four hours by the mouth or a pint by the rectum, at the same interval for twenty-four hours. The disease is so infrequently reported that little opportunity is given for treatment with modern methods such as could be carried out in

the hospital. Just what value there is in the simple remedies above suggested is difficult to say.

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## CHAPTER XXVI

### DISEASES OF THE LIVER, BILIARY TRACT AND PANCREAS

The liver in children is less often affected by disease than in adults. Functional derangements are usually associated with disturbances of the intestinal tract. To such belong the derangement popularly called "biliousness." Icterus of the premature and newly born is usually a physiological process, the explanation of which is not understood. Congenital syphilis is commonly suspected when jaundice occurs in the early months of life, but icterus is not usual in true syphilitic cirrhosis. Umbilical suppurative infections may introduce microorganisms through the portal circulation and cause inflammatory disease of the liver with jaundice, intestinal hemorrhage, high fever, vomiting and diarrhea, ending in general sepsis and death.

In early jaundice which persists and in which the stools have always been light colored, one should think of an obliteration of the bile ducts.

In older children the most common cause for jaundice is the so-called catarrhal form. Chronic and severe jaundice suggests cirrhosis of the liver or obstruction from gall-stones, a very rare disease in childhood. Malignant tumors do occur but are clinical curiosities at this time of life.

**Physiology.**—The functions of the liver as they concern digestion and metabolism are chiefly those of bile formation, and the changing of starch, albumin and fats into glycogen for storage. The liver is also of great importance in the destruction of toxins which may be formed in the intestinal tract, thus preventing digestive disturbances. Microorganisms carried through the portal circulation may be destroyed by the phagocytic action of the liver cells. Bile is highly antiseptic in the intestinal canal, checking putrefaction as well as certain types of fermentation, and the growth of microorganisms.

While the liver at birth is large compared with the size of the body, the functional development is imperfect. The amount of bile secreted and the capacity of the gall-bladder are relatively small in the first few months of life. The growth of the liver proceeds rapidly during each year of life, but not out of proportion to the weight of the body, being about 4 per cent during childhood. In adult life, it is only 2.8 per cent of the body weight.

The normal liver is felt below the costal margin only in the first two years of life. It is soft and highly vascular, however, and for this reason may not be palpable. The upper border in the axillary line is at the sixth interspace.

The characteristics of the minute anatomy of the liver in the young

infant show an irregular outline of the supporting fibers and a less regular arrangement of the liver cells than in the adult. The capillaries are larger and more numerous. Lobules form after one year of age. From the age of eight years, the structure becomes identical with that of adult life.

**Disturbances in Early Life.**—*Absence of Bile Ducts.*—Colorless stools and intense jaundice of the infant from birth may be due to a malformation of the bile ducts. Such a congenital defect is well known, though not common. I have seen two such cases. One infant lived to the age of nine months, and the autopsy revealed the fact that the bile ducts were entirely missing. In other instances, the ducts may be partially occluded. The cause is developmental in fetal life, sometimes ascribed to inflammatory changes *in utero*, but probably of unknown origin. It results in the biliary form of cirrhosis.

The treatment of these cases has been unsuccessful. As the total obstruction of the bile ducts is inimical to life, an exploratory operation is justifiable. It might be possible to connect a patent portion of the biliary tract with the intestine by anastomosis.

It has been suggested that such an infant be fed a soup containing liver emulsion. Liver extracts and bile salts may be tried, but they can do no more than prolong life for a short time.

*Infectious Hepatitis.*—The liver in early life is markedly susceptible to general infection of the infant. A toxic parenchymatous hepatitis or fatty degeneration results from severe infection of any portion of the body, or from intestinal intoxication accompanied by high and protracted fever. Toxins from congenital intestinal obstruction, suppuration of the kidneys (pyelitis of the newly born), infection of the umbilicus, otitis media, furunculosis and abscesses, erysipelas, toxemia of the nursing mother—all result in damage to the liver. Sepsis may cause atrophy of the liver, icterus, or hepatic hemorrhages. Congenital syphilis may, early in life, cause a diffuse hypertrophic cirrhosis.

**Tumors.**—Tumors of the liver at any time of childhood must be considered of great rarity. When congenital, the simple varieties are lymphatic cysts of the ligaments, multiple degenerative cysts and adenomata. Small cysts may cause no symptoms and are harmless. They may be slow in growth through a period of many years. Large cysts cause marked increase in liver dulness and much of the abdominal cavity may be filled. The pleural cavities may be compressed upward by the diaphragm. Parasitic cysts from fetal infestation with the echinococcus have been reported in early life.

Cases of malignant growths of congenital origin have been collected by several writers. Sarcoma may develop independently in the liver or be associated with suprarenal tumor (hypernephroma). Carcinoma, primary in the gall duct, or originating in the liver cells, is of rare occurrence in practice, but the search of the literature reveals the fact that a considerable

number of cases has been reported. I have recently seen a primary hepatic carcinoma in a thirteen-year-old child:

A thirteen-year-old girl developed symptoms four months before admission to hospital. The first evidence was the steadily increasing jaundice. She became constipated, the stools being light colored and finally diarrheal. Bile was present in the urine. The loss of weight was pronounced in spite of the ever-enlarging abdomen, which showed a large liver and free peritoneal fluid. Distention caused pain in the region of the heart. The child easily tired, and the feet and ankles were swollen. The surface of the liver was smooth, hard and

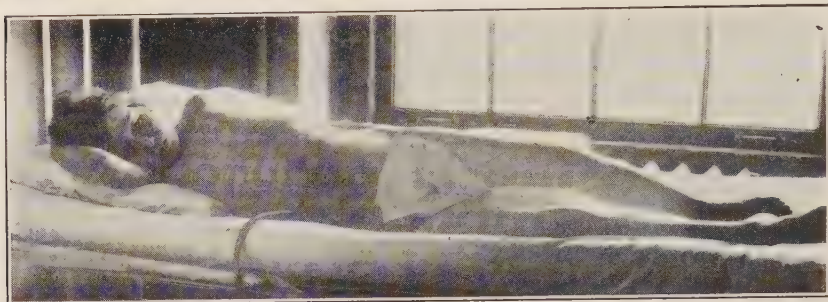


FIG. 52.—PHOTOGRAPH OF THIRTEEN-YEAR-OLD CHILD WITH CARCINOMA OF LIVER  
The abdomen shows sutures and drainage following laparotomy

not sensitive to palpation. Pallor was difficult to determine in the bile-colored lips, but a diminished red blood count and hemoglobin (68 per cent by Sahli method), revealed a definite anemia. The diagnosis was malignancy of the liver. At exploratory operation in the hope of finding an operable obstruction in the gall ducts, a mass was found obstructing the common duct. The child died of malignancy and intercurrent peritonitis, the autopsy revealing a large primary carcinomatous tumor in the right lobe of the liver and a secondary growth in the common duct.

**Catarrhal Jaundice.**—Catarrhal jaundice is a self-limited, acute inflammatory obstruction of the common bile duct, with associated inflammation of the stomach and duodenum. Before the appearance of the jaundice the occurrence of vomiting and constipation lasting several days is suggestive of acute intestinal obstruction. In case the child has been subject to previous vomiting attacks the gastro-intestinal symptoms at the onset may be mistaken for cyclic vomiting. After three or four days the physician is surprised to find a rapidly developing icterus. In childhood this sequence of symptoms is seldom found belonging to any other disease than acute catarrhal jaundice.

Of chief interest is the seasonal incidence. Most cases occur in the fall months at a time when epidemic respiratory diseases are present. It is therefore best explained upon the basis of infection. Some cases develop without a stormy onset, and the cause is determined with difficulty, if at all. The mode of transmission may be hematogenous from the respiratory tract.



More than one child in the family may be affected in the course of weeks. The disease is rare in infancy, and is uncommon between three and six years.

The disease usually begins as a gastroduodenitis, to be followed soon by inflammation of the mucous lining of the bile duct. From the obstruction caused thereby little if any bile reaches the intestinal contents and the stools are gray or white. The skin, mucous membrane and urine contain much bile. When the blood is obtained in the test-tube and the clot allowed to form, the serum will show the dark red bile-tint. Testing of the blood serum with the Van den Bergh reagents gives an immediate reaction indicative of biliary obstruction.

The following case history will portray the symptoms and course as I have frequently found them:

A girl of eleven years vomited persistently for three days. No bowel movement had occurred. There were abdominal distention and tenderness in the region of the stomach, duodenum and gall-bladder. The liver was enlarged. Severe prostration and dehydration had developed. The appetite was gone and the tongue was heavily coated. She complained of headache. The appearance was one of lassitude and there were usually one or two degrees of fever. On the fourth day, the conjunctiva and skin were found to be definitely jaundiced. The urine became dark with a yellow foam when shaken. The stools were dry and grayish-white. In the beginning of the second week, the appetite improved and local symptoms in the abdomen disappeared. Jaundice had entirely faded after two weeks from the onset.

*Treatment.*—Some children are not confined to bed and may continue in school. Others with a stormy course are prostrated. Rest in bed is essential for these. The dietary treatment is chiefly important, fats and milk being restricted. Lavage with soda bicarbonate solution is of value for persistent vomiting, and phosphate of soda for the constipation. Plenty of water should be given, and as soon as the stomach is tolerant, fruit juices in large amounts will be found to increase the appetite and help to supply the necessary fluids.

**Hepatic Insufficiency.**—Gastro-intestinal disturbances with toxemia in the young child may produce an insufficiency of the hepatic functions with a reduction in bile. Children may complain of headache, the skin is sallow and suggests mild jaundice, the stools are dry and pale, the urine reddish. The tongue is dry. There may be nosebleed, and hemorrhage from the stomach. Mild fever, variable appetite, and emaciation are usually present. The liver is not palpable. The general condition resembles tuberculosis, but with rest in bed, a fruit diet, and daily enemata the child recovers. The cause is a simple toxic disturbance of the liver tubules, secondary to intestinal toxemia.

*"Bilious Attacks."*—One still hears the term "biliousness" used by the laity. It has no place in medical terminology, as it does not belong to any definite diseased condition. Long popular usage has coupled it with gastro-



intestinal upsets of poor appetite, vomiting, constipation, headache and dizziness, and sallowness of the skin, all of which are assumed to be due to a disturbed function of the liver. When once a child has had such a syndrome, all succeeding illnesses are apt to be classed as recurrences of "biliousness." In all such cases a careful physical examination will reveal the true condition, and this may be one of many ailments to which children are susceptible. All of the symptoms just mentioned may be found in chronic infections of any portion of the body. If any one disease might be considered as "biliousness" it is hepatic insufficiency. I have known many diseases from diphtheria to appendicitis to be neglected while the parents were resting under the supposition of "biliousness." Carious teeth, infected tonsils, disease of the mastoid and nasal sinuses, bronchial fibrosis, tuberculous glands and pyelitis are particularly liable to be so mistaken. Less serious conditions that fall into this category are constipation from one-sided diets or from neglect, and insufficient sleep and rest. The important point for the physician to remember is that the symptoms commonly ascribed to this popular disturbance may be the manifestations of a serious disease which needs to be promptly recognized and properly treated.

**Benign Jaundice from Round-Worms.**—Round-worm infestation of the bile ducts and even of the liver has become proven. The *Ascaris lumbricoides* has been discovered accidentally at abdominal operation. In other cases the ova have been found in the stools of individuals suffering from jaundice and other symptoms of biliary obstruction, and at operation the parasites recovered when the bile ducts were opened. It may be difficult to find the eggs in the stools if the worms are present only in the biliary passages. In some reported cases expulsion of the worms either spontaneously or as a result of anthelmintics cured the jaundice.

Pain in the region of the ducts is not always found, nor is fever a necessary accompaniment. The symptoms may be only those of obstructive jaundice, with icterus of the skin and conjunctiva, clay-colored stools and bile-colored urine. The child may be nauseated and dizzy.

Inflammation of the bile ducts results from the irritation caused by the parasites, and without treatment may lead to serious results and death. Vigorous medication with santonin and calomel will result in expulsion of the worms and cure in some cases, but laparotomy is justifiable and necessary when other treatment is unsuccessful.

While such cases are rare in this country, persistent jaundice should be considered as possibly due to this cause.

**Gall-Stones (Cholelithiasis)** and gall-bladder disease (cholecystitis) are rare in childhood, but there have been collected as many as eighty cases. The disease may not be so unusual as it is undiagnosed. In children the disease is more common in males, a marked difference from the adult female preponderance. Small gall-stones are not infrequently found in infants at autopsy.

The history of typhoid fever is obtained in some cases, but the etiology points in some instances to bacteremia from coexistent appendicitis or scarlet fever. Cultures taken from the gall-bladder at the time of operation may show pyogenic cocci and colon bacilli in mixed infection.

The symptoms so nearly resemble appendicitis that the preoperative diagnosis may be difficult. Appendicitis often accompanies the gall-bladder disease. The history of recurrent pain should make one conclude that gall-stone disease rather than appendicitis is present. Sudden attacks at irregular intervals may keep the child bedfast for a few days. Jaundice usually appears previously or during the attack. A palpable mass in the region of the gall-bladder can be detected. The liver may be larger than normal. The general symptoms resemble those of adult life. They consist of vomiting and pain in the right side of abdomen or near the umbilicus. Tenderness near the region of the gall-bladder is elicited by palpation under deep inspiration. Rigidity is present in the right rectus muscle. Posteriorly tenderness is found at the right of the tenth and eleventh dorsal vertebræ. There is only a moderate increase in the leukocytes, but the differential count shows a high percentage of polymorphonuclears. The urine contains bile. In severe cases there will be albumin and casts, due to toxemia and the resulting renal degeneration.

The three symptoms, jaundice, a mass in the right upper quadrant and recurrent pain present a syndrome belonging to typical cases of gall-bladder disease. Any one or all of these, however, may be absent in a given case, and the condition only discovered at operation.

While gall-stones in children may never be encountered in a lifetime of practice, one should remember that symptoms resembling those of stones may occur, probably due to infection of the biliary tract and retention of bile. In such cases there will be colic in the umbilical and upper abdominal region, poor appetite and obstinate constipation. It occurs in the obese child, and there is a history of susceptibility to general infections. It is probable that the cause is hereditary. Such children should have a reduction in the fat intake, and placed upon a diet containing plenty of fruit and green vegetables.

Congenital narrowing of the cystic duct may result eventually in gall-bladder inflammation and the formation of stones. In such cases there will be recurrent pain in the upper abdomen, cramps and diarrhea. During acute attacks vomiting occurs.

At operation the gall-bladder is usually found enlarged and distended with evidence of much inflammation. The contents consist of pus and thickened bile. Stones may be found in the gall-bladder without the presence of pus. Calculi of various sizes are found, and some as large as an inch in diameter have been removed from children.

The gall-bladder should be inspected in all cases where the abdomen is

opened because of abdominal pain, even if the appendix is regarded as pathological.

The prognosis depends upon surgical relief by drainage. The child who has not been weakened by the long duration of the disease offers a better outlook and operation results in cure. Otherwise the child will probably die of sepsis.

**Cirrhosis.**—It is not the purpose of this volume to discuss the subject of the cirrheses of the liver, but simply to mention the causes which produce the types usually found in early life.

Many children dying from gastro-intestinal diseases show at autopsy secondary inflammatory or degenerative changes in the liver cells.

In childhood cirrhosis is due to malformations of the bile ducts, to syphilis and to infectious inflammation. An infantile type of biliary cirrhosis is reported in India and Mexico, appearing as a family tendency. Formerly cirrhosis was ascribed to the influence of a faulty diet and digestion, but the influence of infection is now recognized as the etiological factor even if the source is not always found. The finding of a marked leukocytosis mostly of the neutrophilic type favors such an origin. The early inflammatory process is characterized by the destruction of liver cells. Death may occur before the later changes of cirrhosis can develop. Following the inflammation of the liver, there appears in less acute or severe infections, attempts at regeneration of the liver cells and the formation of new bile channels with fibrosis formation (hardening, cirrhosis). Cases associated with enlarged spleen are probably identical with Banti's disease (splenomegaly with cirrhosis of the liver).

Toxic cirrhosis, due to poisoning from alcohol or lead, is a rarity in children in this country. Twins reported by Ely developed cirrhosis following the feeding of brandy for several years.

Congenital syphilis in infants may cause an intercellular cirrhosis, not always accompanied by enlargement of the liver. The gross appearance of the liver may be normal, though in some cases the cut surface of the liver may show millet-seed gummata. Jaundice is not common. The clinical diagnosis of syphilis is made by signs elsewhere in the body, and not by symptoms in the liver during life.

The treatment of all forms of cirrhosis is unavailing.

**Fatty Disease of the Liver.**—At autopsy, enlargement of the liver and a "buttery" color are frequently observed as a result of protracted or severe toxemia of bacterial origin, or from chemical poisoning. The changes are due to fatty degeneration or infiltration. During life the edge of the liver is palpable below the costal margin and the consistency hard. There are no other symptoms referable to the liver, and the diagnosis and treatment depend upon the recognition of the underlying infections or toxic cause.

**Hydatid Disease.**—This disease, rare in children, is due to transmission through the bile ducts of the larvæ of the small or dog tapeworm, *Tænia*



*echinococcus*, with the formation of cysts in the liver. As a result the liver becomes greatly enlarged, and fluctuation may be detected. Pressure symptoms are the chief manifestations of this cystic disease. The diagnosis is confirmed by the aspiration of the infected fluid contents of the cysts. Suppuration, sepsis, spontaneous rupture of the cyst may occur and make the outcome serious and usually fatal. The early diagnosis and surgical treatment are necessary for saving life. Removal of the cysts with complete recovery has been reported in children.

**Diseases of the Pancreas.**—The recent great advances made in the knowledge and treatment of diabetes mellitus have confirmed the earlier belief that the normal pancreas produces an internal secretion which regulates the metabolism of carbohydrates. This function belongs to the masses of cells placed in the interstitial tissue of the pancreas and known as islands of Langerhans. It is the destruction or disturbance of these islands which interferes with the feeding of starches and sugars, and results in diabetes. The routine examination of the urine has revealed the fact that this disease is not so rare in children as formerly supposed, being by far the most frequent disturbance of the pancreas. Insulin is becoming as widely used in children as in adults, and has greatly improved the prognosis as to life, but there still remains the problem of insufficient body growth and height which are not entirely corrected by the use of this agent. As this disease belongs to the domain of metabolic disturbances and is treated in another volume, the subject is not to be further considered here.

The other physiological functions of the pancreas are directly concerned in the digestive processes. When the pancreatic secretion reaches the common duct and the duodenum, it becomes activated by intimate mixture with the bile and intestinal secretions. Starch digestion is completed by the ferment amylopsin, protein by the action of trypsin and fat is split by the combined action of steapsin and bile. While all these ferments are present at birth, they are increased in quantity as the child grows older. This is easily understood when one considers the rapid growth in the pancreas, for the weight doubles at five months of age, grows steadily until the end of the second year, when during the following year it again doubles its weight.

The nature of the disturbances in the pancreas and the rôle they may play in the intestinal diseases are not yet determined. The secretion of the pancreatic ferments is probably greatly lowered in all febrile diseases, especially those of gastro-intestinal origin. Fats and carbohydrates will not be completely digested in the usual amounts taken, when the intestinal juices and the bile are insufficient to activate the pancreatic ferments which have entered the bowel. Insufficiency of the pancreatic function has been suggested as the explanation of chronic fatty diarrhea and this seems plausible clinically, but has not been proven by any changes found in the pancreas at autopsy. The best known clinical disturbance of fat and carbohydrate tolerance is that known as celiac disease, considered as a form of chronic



intestinal indigestion, for want of a better knowledge as to its causation. In practice one finds that the amount of these foods must be greatly reduced, not only in these cases, but in all disturbances of childhood accompanied by fever and undigested stools.

*Disease of the Pancreas.*—In children demonstrable disease of the pancreas is rare, and is then usually secondary to processes elsewhere in the body. The most common pathological changes found at autopsy are the miliary lesions of generalized tuberculosis. Hereditary syphilis of the active and severe type may involve this organ with the production of small gummata, multiple hemorrhages, atrophy of the secretory cells, or the production of a chronic inflammation and resulting hardening (fibrosis). Acute hemorrhagic pancreatitis is not known in children.

Parasitic invasion occasionally occurs, probably more often than suspected, because of the ease with which intestinal worms may travel up through the ducts. One observer reported three cases of pancreatitis due to the irritation of round-worms in the pancreatic duct. Hydatid cysts may likewise develop in the pancreas, though not so often as in the liver.

Malignant tumor of the pancreas in children is rarely observed. Sarcoma has been reported. Retention cysts have resulted from severe trauma of the pancreas. A large tumor of the pancreas will show a change in the edge of the gastric shadow seen in the barium radiogram.

Acute suppurative pancreatitis may result from penetrating wounds, or from the extension of a pyogenic focus in general sepsis. Acute inflammation may occur in children during or after such acute infections as typhoid fever, measles, chickenpox, scarlet fever or diphtheria. Infection may take place through the blood stream, but it is possible for microorganisms to pass from the intestine through the pancreatic ducts and cause inflammation thereof. Acute pancreatitis is a rapidly fatal disease, death occurring usually within the first twenty-four hours. It is probable that the pathological change in the pancreas is an autodigestion of its tissues, due to the change in the ferments produced by the toxins of the infection.

Quite a number of observers have recorded the occurrence of a pancreatic disturbance during or following the course of mumps. One writer called the manifestation "mumps of the pancreas." It is a mild and temporary affection, and if there actually occurs an inflammation of the organ, it is far less severe and of a different type than acute pancreatitis from other causes. I have seen one case, that of a ten-year-old boy, who at one week from the beginning of parotitis developed the characteristic secondary rise of temperature, repeated vomiting, dizziness, malaise and increased urinary frequency and amount. The leukocyte count was normal. In addition to these symptoms, abdominal disturbances are usually present, consisting of pain and tenderness in the region of the pancreas, and abnormal stools, either constipated or showing a fatty diarrhea. Sugar has been found in the urine of some cases. It has been noted that the pulse is abnormally slow, dis-

tinguishing this type of pancreatitis from the severe form. The disturbance lasts from two to ten days and is followed by complete recovery. No treatment is necessary other than complete rest in bed and the use of a diet low in fat and sugar. The theory, not proven, has been offered that inflammation from this cause may produce fibrosis and permanent disturbance in function resulting in diabetes. No operative or postmortem report on the tissue change has been made so far as I know, nor has any one noted diabetes following mumps.

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## APPENDIX

### TECHNICAL METHODS IN PEDIATRIC PRACTICE

**Roentgen Ray in Gastro-intestinal Diagnosis.**—The use of the roentgen ray is of great value in children and as necessary for correct diagnosis in many cases as in adults. An opaque medium given by the mouth or rectum is indicated in certain cases. In the examination of the digestive tract both the fluoroscopic screen and the radiogram are useful. In many instances the roentgen picture gives us the first clue as to the diagnosis; in others it confirms the findings. The clinician should not be misled by x-ray reports which are at variance with well-defined clinical symptoms. Probably the roentgen examination is not made so frequently as it should be, for if it discloses no pathological conditions, it is of value in their exclusion.

In general one should use the fluoroscope for determining the function of the stomach and intestines as regards the motility and the nature of the contents. It has taught us much about the presence of air in the stomach. When the infant is recumbent the fundus, which is chiefly in front of the esophageal opening, is filled with air, while the food contents cover the upper opening and prevent its escape. In the upright position, the fluid sinks and the air reaches the esophageal outlet. This mechanism has much to do with the comfort and appetite. Such observations have shown that feeding too slowly and in the dorsal position causes more air to be swallowed and it is retained unless, by a shifting of position or by vomiting, the gas escapes. After feeding with a full meal, the distal portion of the contents is seen entering the pylorus, but an overdistended and dilated stomach cannot empty promptly, and there results a delay in evacuation and a considerable residue. The fluoroscope enables one to study the presence and rapidity of peristalsis, especially the change in the outline of organs when the abdomen is palpated.

The shape, size and location of the abdominal organs are roughly determined by the radiographic film. In this way a permanent record is made which may be useful for later comparison.

The physician should not be content with sending the child to the roentgen laboratory and depending alone on a written report, but he should accompany the patient, watch the phenomena which are presented by the screen, and discuss the symptoms and findings with the roentgenologist. He should become able to interpret for himself the disclosures of the fluoroscope and radiogram and correlate these with the clinical manifestations.



The most common diagnostic employment of the roentgen ray in children is for the location of foreign bodies; and unfortunately it is not used enough. These are sometimes found accidentally during routine fluoroscopy if the object is opaque enough to cast a shadow. Anywhere from the esophagus to the rectum the foreign object can usually be visualized either from its own density or by a barium coating.

In acute diseases due to inflammatory obstruction, the diagnosis may be sufficiently clear from the symptoms, so that one is not always warranted in the delay, or the tax upon the child's strength attendant upon a roentgen examination: acute intussusception illustrates this fact. Here the presence of early signs of abdominal pain, vomiting, collapse and blood in the stool is sufficient for a diagnosis. In appendicitis in children, the roentgen-ray examination is of no benefit. In cases with atypical symptoms, the appendix may be filled by parasites or fecal material. In such a case or if there be adhesions, the appendix cannot be visualized. Sometimes normal appendices do not fill with barium.

In pyloric stenosis the diagnosis of a typical case is sufficiently clear without a radiogram. The clinical picture is characteristic. Delay in the emptying through the pylorus may be due, however, to spasm of the muscle or to a lack of tone in the gastric wall. Here the barium will eventually pass into the intestine, and the radiogram enables one to exclude a complete or surgical obstruction. The fluoroscope may be used for studying the rate at which the stomach empties. When the pylorus is patent, the barium is seen entering the duodenum even while the meal is being taken. The failure to begin emptying at once and the presence of most of the opaque substance in the stomach four hours later is a valuable confirmation of pyloric obstruction. When the condition of the infant warrants delay, a radiogram at twenty-four hours is of value. When the obstruction is complete, there will be found a shadow in the stomach representing the barium, and little or none in the intestines at the end of twenty-four hours.

In congenital obstruction of the esophagus, the immediate vomiting of all fluids taken by the newly born infant with failure to permit the passage of a catheter (not over No. 12 French) is all that is needed for the diagnosis of a complete stenosis. But the outline of a blind ending to the esophagus, or a narrow shadow caused by a diverticulum or a partial stenosis discloses the nature and location of the abnormality.

Intestinal obstructions of congenital origin are diagnosed with certainty by the opaque media. This method should never be omitted when vomiting and absence of stools occur, as it gives an indication of the site. The duodenum, jejuno-ileum junction, and the rectum are the most frequent locations. Meckel's diverticulum is not often disclosed, but the stasis at the level of this malformation gives some indication of the region of obstruction.

In intussusception, the barium enema will rise to the lower level of the obstruction, and can be seen under the fluoroscope, passing the sigmoid and

descending colon to the point of obstruction, where the barium shadow ends abruptly or traverses the invaginated bowel as a thin line. A routine examination when the symptoms first appear would be a valuable time-saver and permit a surgical diagnosis, with cure of the child, much sooner than usually occurs. Barium injections are not necessary or advisable in acute intussusception, but help in the diagnosis of chronic invagination.

The location of intestinal or abdominal tumors causing obstruction is shown by the barium contents. Radiography is of value in cases of fecal impaction and intestinal stone. Redundancies, kinks and the presence or absence of haustra in the intestine are disclosed by this method. Calcareous mesenteric glands give a typical mulberry appearance, and are usually seen in the right lower abdominal region. The shadow becomes more evident as the calcification increases.

Dilatation of the colon is clearly shown. It does not enable one to make a diagnosis of Hirschsprung's deformity or celiac disease, but it is suggestive.

The abnormal position of the hollow viscera in the abdomen is well determined by the roentgen ray, and is of practical value in estimating the degree of dilatation and ptosis. Transposition of the viscera from one side of the abdomen to the other or through a hernial opening in the diaphragm is discovered by the radiogram. Not infrequently one sees abnormal upward bulging of a portion of the diaphragm which represents a defect in its integrity. In true diaphragmatic hernia, the presence of dextrocardia or of thoracic tympany may be sufficient to fix the diagnosis, but often the fluoroscope gives the first indication that the stomach or intestine is within the thoracic cavity.

Peptic ulcers of the gastro-intestinal tract are uncommon in childhood, but their recognition by the radiogram would be of much prognostic value in that operation before perforation would be made possible. Gastric ulcer has been diagnosed in later childhood by the presence of a crater filled with barium, giving a shadow projecting outward from the normal outline of the lesser curvature of the stomach. In rare instances it has been possible to show the findings of duodenal ulcer similar to those in adults. In the rather rare disease of chronic ulcerative colitis, the radiogram shows narrowing of the lumen of the gut, and the absence of haustra.

**Thick Feeding.**—The food of young children is often too greatly diluted. The recent tendency of pediatricians is to give more concentrated feedings, especially in those infants who take food unwillingly and in amounts insufficient to cause gain in weight and growth. Thick food is of value in the difficult feeding of infants with cleft-palate. In most cases of vomiting, whether from rumination, air-swallowing, deficiency of the size or tone of the stomach, hypertrophy of the pylorus, gastro-intestinal spasm (so-called nervous vomiting), or dyspepsia in the breast-milk infant, some of the food should be thick, and it will be found to be better retained than if thin. When the child is living on a concentrated diet, it is essential that

water be administered between feedings, so as to supply the fluid needs.

The milk may be concentrated or thickened by the simple methods of boiling, souring or peptonizing. Cereals may be cooked with milk rather than with water. Powdered milk readily adapts itself to thick concentrated solutions. With the child who is getting plenty of water in other ways, there is no object in diluting the orange juice, in giving thin soups or broths or in straining well-cooked cereals and vegetables. Gelatin is a good addition to milk, broths and juices, making them thick or stiff, and adding to their nutritional value. It is of benefit especially in vomiting and diarrhea.

**Sour Milk Feeding as a Therapeutic Measure.**—There is no doubt about the value of sour milk in feeding either normal or sick children. Buttermilk has been used since 1770, and in the past twenty-five years, especially in Holland and Germany, its value in diarrhea has been proven, and its use attained popularity. The presence of lactic acid bacilli has been thought to have a beneficial action by the influence upon the intestinal flora. The acid prevents an abnormal proliferation of bacteria. Marriott found that the addition of lactic acid to cow's milk increased its digestibility to that of breast milk. Gastric digestion occurs best when a constant acid reaction is present in the contents. Undiluted whole sweet milk has an unfavorable influence upon the gastric acidity of very young infants, whereby the protein splitting by the pepsin is hindered. Acidulated milk can be successfully fed to young infants even in the newly born period. Milk, soured either with the organisms or with the U.S.P. acid, is of use in constipated or diarrheal infants.

Three of the commonly used milks and the method of souring are :

Hydrochloric acid milk as devised by Faber is prepared by adding 100 c.c. ( $3\frac{1}{3}$  oz.) of 1 : 10 normal hydrochloric acid to each pint (450 c.c.) of boiled whole sweet milk.

Lactic acid milk may be prepared by souring with buttermilk tablets or culture, as has been the custom for the past fifteen years, or by the method of Marriott, using less acid than he originally suggested : to each 500 c.c. (approximately 1 pint) of milk, add 2 c.c. ( $\frac{1}{2}$  drachm) U.S.P. lactic acid.

Citric acid (lemon juice) milk as suggested by Hess is prepared as follows : to each liter (approximately 1 quart) of milk, add 21 c.c. (5 drachms) undiluted lemon juice.

Buttermilk, naturally soured, or with the lactic acid organisms, has been found to be well tolerated and answers all the requirements for successful feeding as regards the physiological digestion. It varies markedly in its acidity by aging, and in hot weather. The more recent methods of souring are based on scientific determination, and produce a quality of milk which is probably more pleasant to the taste.

**Tube Feeding (Gavage).**—With a No. 16 French soft rubber catheter introduced only as far as the lower third of the esophagus (6 inches in



the infant), liquid food is injected from a one-ounce Luer or larger glass syringe. It expedites the feeding to have a second syringe loading while the first is being emptied; or the piston may be discarded and the food poured from a pitcher or beaker. I have found the commonly used funnel to be slower and to complicate the emptying of the catheter through obstruction from air bubbles. Care should be taken that air be not forced into the stomach.

**Methods of Water Administration.**—The use of plenty of water is now recognized as one of our most useful therapeutic agents, and one which is sometimes neglected. Throughout this volume many references have been made to the indications for plentiful amounts of fluid in the diseases characterized by vomiting, severe diarrhea, high fever, and other conditions in which there is a drying-out of the body. As a rule no other method can compare with the oral administration. Obviously this is futile in persistent vomiting, and impossible when for any reason the child will not or cannot drink. During the first days of high fever, thirst is extreme, but this may be replaced by a disinclination to drink. The physician often sees cases in which the administration of water has been neglected. When the urgency for increased fluids is great, more than one method of giving them is advisable.

As lavage is indicated in severe vomiting, it is well to follow it by leaving a sufficient amount of water in the stomach. This should be done daily as long as necessary.

Water is given by the bowel more frequently than by any other of the unusual ways. It is certainly to be recommended as a valuable method, and is the most practical in the home.

Hypodermoclysis is usually more applicable to hospital use, but there is no reason why the physician should not use it in the home. It is the simplest and most serviceable of all the parenteral routes for supplying water.

In desperate cases the occasional intravenous administration of watery solutions is beneficial, but seldom used in children because of the difficulty of retaining the needle in the vein during administration, and the fear of reactions. It is applicable only in hospital practice.

The intraperitoneal route has come into use because of the facility with which large amounts of fluid can be introduced. I have frequently employed it during the past eight years since it was introduced into this country by Howland, Blackfan and others.

**Stomach Washing (Lavage).**—The prompt washing out of the stomach is indicated after the swallowing of irritant drugs, poisons, contaminated food or foreign substances which can be removed in this way. The common practice is to add a drachm of bicarbonate of soda to the pint of water, and for the pronounced vomiting of toxic origin this is advisable. In repetition of lavage several times daily, it is advisable to use plain water



only, as the constant neutralization of the acid gastric juice for an extended period is unwise in childhood as severe alkalosis can be produced.

Lavage is usually a simple procedure, but the physician should familiarize himself with the technic in babies and in older and struggling children. The funnel-catheter improvised apparatus is usually at hand. This method is somewhat slower than necessary. A more rapid and satisfactory apparatus is the large syringe holding an ounce, and having a long nozzle which is easily attached to the catheter. A new catheter is to be preferred, in size from a No. 16 to 24 French. Infants never require smaller than a No. 16. One syringe after another is emptied through the catheter, the upper end of the catheter being pinched while the syringe is being loaded, and released from time to time to allow the gastric contents to escape. The child is held on the table with the head drawn to one side. The cheek is pushed between the teeth to prevent biting of the catheter or the operator's finger.

**Proctoclysis.**—Watery solutions of salt and glucose are sometimes retained and absorbed from the lower bowel, when inflammation and straining are not present. If the child's coöperation can be obtained and the buttocks be compressed tightly, the procedure will be more successful. The services of an experienced nurse should be obtained, unless the physician himself desires to give the treatment. Nutrient enemata such as milk and eggs are not well borne, nor absorbed, by children.

Physiological solutions of sodium chlorid, or glucose from 5 to 15 per cent in water, are best given slowly, by the drip method, the quantity being suited to the needs and tolerance of the individual child. The lower bowel is first emptied of fecal matter, followed by the introduction of a firm catheter, preferably No. 20 French, well lubricated. It should be passed slowly until it reaches the sigmoid, where it is to remain. The container should be held not over a foot above the child's body, and the flow regulated so that an hour or more is required for introducing the amount desired.

Proctoclysis is indicated when vomiting, insufficient drinking, or dehydration has occurred. Fluids can be repeatedly given in this way, but not oftener than once daily, because of the local irritation resulting from more frequent injections. Occasionally a sick child, bordering on shock, will not stand this treatment, and may show collapse. Lack of success in performing proctoclysis calls for some other method of fluid administration.

**Hypodermoclysis.**—The hypodermic administration of considerable amounts of physiological salt solution is well tolerated in children and should be used more commonly in replacing water lost by protracted vomiting, diarrhea, and high fever. It is of value as a febrifuge. Sugar solutions should be filtered through gauze, sterilized, and be not over 3 per cent strength if injected by this method, and never used in emaciated subjects because of sloughing or failure of absorption. The injection of solutions of bicarbonate of soda should not be attempted as they are irritant to the subcutaneous tissue.

Most hospitals keep an autoclaved apparatus and solution ready for use, and this may usually be secured for use in the home. A gravity apparatus is required, such as an irrigation bottle or enamel can, to which can be attached a rubber tube several feet long, ending in a "Y" glass connector with double terminal tubes and two needles. The temperature of the water in the container should be kept about 105° F. From 1 to 2 pints will be taken up by the tissues in one or two hours, repeated daily if indicated.

The injection should be given in a region which has much loose subcutaneous tissue, and in which the discomfort will be the least. As the child usually lies upon the back, and injections result in a sensitive area for a day or two, it is better to select a site on the anterior portion of the body, over the abdomen, lower part of the chest or sides of the thighs.

**Intraperitoneal Injection.**—For the relief of acute dehydration, the intraperitoneal injection of salt solution is practically always followed by some benefit and sometimes by striking improvement. When the child's circulation is good, absorption takes place readily. The restlessness, the shrunken appearance of the face and abdomen, the weak pulse and the scanty urine soon begin to return towards the normal. It is necessary that the cases for intraperitoneal injection be properly selected, and that the technic as regards the introduction of the needle and the quantity of water be safeguarded by a large degree of care and experience.

The technic should be aseptic, the needle should have a short but sharp bevel, the bladder should be empty. The median line just below or the region to one side of the umbilicus outside the rectus muscle is the site usually selected. The skin is cleansed with iodine and alcohol. The needle is introduced at an oblique angle, through a portion of the abdominal wall which is held between the fingers and away from the intestines. The needle is pushed carefully through until the peritoneum has been pierced. The solution is then allowed to flow from a funnel or gravity bottle. Three ounces can be safely used in infants, 6 in medium-sized children, and 8 in larger children. The fluids which seem to me to be the safest are distilled water, isotonic salt solution, and Ringer's solution. Glucose, when not stronger than 5 per cent, is well absorbed by the peritoneum in children who are not in collapse. Concentrations of 10 to 15 per cent should not be given by the peritoneal route.

*Discussion.*—This method is not without danger. To my knowledge an accidental needle puncture of the intestine happened in a child in a neighboring city, but I do not know the circumstances leading up to the catastrophe. One should never try to puncture the peritoneum if there is marked abdominal distention, or if the intestine should be adhered to the peritoneum at the site selected.

The quantity and the concentration of the solution to be injected should not be too great. In the presence of a failing circulation this method should not be used. At the Kansas University Hospital lack of absorption was

noticed in two infants who had been severely ill with ileocolitis. Five per cent glucose was given to one and caused no improvement. At autopsy all the quantity injected was found in the peritoneal cavity. In the second instance the injection of glucose caused so much discomfort and distention that the house physician reintroduced the needle, withdrawing more fluid than had been introduced.

In marasmus and terminal infection, such solutions are not well absorbed either by the peritoneum or subcutaneously. This is due to the fact that osmosis is in the direction of the fluid having the highest tension.

As a practical method of determining the safety of intraperitoneal injection, one should see that the pulse is strong enough, and that the blood pressure is not abnormally low. Salt solution should be isotonic; that is, it should have the same osmotic pressure as the blood. Five per cent glucose solution is the highest limit of safety, and further experience may show that it should not be used at all. Children who have already suffered much water loss will only be further damaged by abstraction of water from the circulation and its exudation into the peritoneal cavity.

**Drug Dosage for Children.**—In estimating the size of the dose which is to be given, experience is the best guide. With such drugs as atropin, the child takes a relatively larger dose than is usually given to the adult. In progressive postdiphtheritic paralysis, for instance, strychnin is given to a two-year-old infant in gradually increasing dosage until  $\frac{1}{60}$  or  $\frac{1}{30}$  of a grain may be reached per dose. Such a large dose naturally would not be given as a tonic or for ordinary therapeutics. With a few drugs such as morphin, which is to be given in relatively smaller doses and with great caution, the physician feels more comfortable if he follows a mathematical rule in estimating the size of the dose to be given.

The two most common formulæ are:

1. Multiply the age by 5, and the product is the percentage desired. For instance, age 2 years times 5 equals 10 per cent of the adult dose.

2. Add 12 to the age and divide by the age. For instance, age 2 plus 12 equals 14, divided by 2 equals 7. The child should be given  $\frac{1}{7}$  of the adult dose.

At two years of age the percentage of the adult dose is 10 by the first method, and 14 by the second. This variation may be disregarded usually, although it may be adapted to the comparative weight of children, using the smaller dosage for light weight children and the larger for the heavier.

**Widal Reaction.**—Because of the helpfulness of a positive blood test in the diagnosis of typhoid fever in children, one may be pardoned for repeating here something about the nature of the reaction. The details of performing the test may be found in nearly every book on laboratory diagnosis.

There is formed in the blood of typhoid patients certain protective sub-



stances capable of inactivating or agglutinating the typhoid bacilli. The presence of this protective substance (agglutinin) can be determined by placing a small amount of the patient's serum in contact with a dilution of typhoid organisms. If agglutinin be present, the organisms will form in clumps and remain motionless, and if no agglutinin exist the organisms remain separated and show motility.

The amount of agglutinin in the patient's blood varies. The serum should be diluted so that it represents proportions of 1 : 20, 1 : 30, 1 : 50, or higher. The test is more valuable if the high dilutions such as 1 : 50 bring about a complete loss of movement and a marked clumping of the organisms within an hour. Some normal bloods may have slight agglutinating power and it is only with the higher dilutions that importance is attached to the agglutinating phenomenon. The rapidity with which the reaction takes place is also important. The very low dilutions of serum, containing much agglutinin, will bring about clumping much faster than a high dilution. The reaction is carried out in small test-tubes, read macroscopically or with a few drops of the serum dilution and the typhoid suspension on a hanging drop slide under the microscope.

**De Silvestri's Color Reaction in Typhoid Urine.**—This reaction is useful in the early stage of typhoid and paratyphoid before the blood gives the typical agglutination tests of Widal. It has been used by the writer for several years and found of considerable value.

Heat the urine and if it show a precipitate of albumin or phosphates, this should be filtered out. Place 2 c.c. of tincture of iron chlorid in a test-tube, add 5 drops of pure sulphuric acid and carefully superimpose 3 c.c. of the suspected urine. At the point of contact a brownish-yellow ring appears and spreads upward into the urine. A ring of cloudiness with greenish reflections is seen on the surface of the urine but disappears when the test tube is shaken. The brownish-yellow or chestnut tint however remains after shaking or heating the test-tube. This is the characteristic finding in the urine.

**Test for Occult Blood in the Urine and Stool.**—When blood is not visible to the naked eye, its presence may be easily determined by the following method, which has been used for years in my practice:

Take 5 c.c. of urine and add 1 drop of acetic acid and then add 5 c.c. of Meyer's solution. With a pipet overlay this mixture with 2 c.c. of peroxid. At the line of contact a bright red ring appears if blood be present in the urine.

For determination of blood in the stools a similar method is used. A small portion of fecal matter from the center of the stool is added to 5 c.c. of water to which has been added a drop of acetic acid. Meyer's solution and peroxid are added as described in the examination of the urine, and a bright red ring is characteristic of blood.





## INDEX

- Abdomen, causes of obscure pain in, 269
  - examination of, 267
- Abdominal purpura, diagnosis of, 274
  - symptoms of, 273
  - treatment of, 274
- Abdominal support, cyclic vomiting treated by, 138
- Abdominal tuberculosis, complications of, 283
  - diagnosis of, 282
  - etiology of, 277
  - general survey of, 276
  - intestinal lesions of, 279
  - mesenteric lesions of, 280
  - occurrence of, 277
  - pathogenesis of, 277
  - peritoneal lesions of, 280
  - prognosis of, 284
  - symptoms of, 280
  - treatment of, 284
- Abscess, gingival, 13
  - peritonsillar, symptoms and treatment of, 42
  - retropharyngeal, diagnosis of, 44
  - — symptoms of, 43
  - — treatment of, 45
  - typhoid bacilli causing, 186
- Accidental poisoning, acids, 311
  - alcohol, 311
  - alkalies, 310
  - arsenic, 311
  - aspidium, 312
  - atropin, 312
  - camphor, 312
  - chenopodium, 312
  - formaldehyd, 313
  - lead, 313
  - mercury, 313
  - opium, 313
  - phosphorus, 314
  - santonin, 314
  - silver nitrate, 314
  - turpentine, 314
- Acid, accidental poisoning with, 311
- Acrodynia, teeth and, 64
- Actinomycosis, pharyngeal, 40
- Acute appendicitis, affections mistaken for, 254
  - anatomy of, 250
  - diagnosis of, 253
  - etiology of, 249
  - pathology of, 250
  - prognosis of, 255
  - symptoms of, 251
  - treatment of, 255
- Acute catarrhal gastritis, pathology of, 104
  - symptoms of, 105
  - treatment of, 105
- Acute catarrhal pharyngitis, treatment of, 39
- Acute dysentery, course of lesions of, 175
  - description of, 173
  - diagnosis of, 178
  - etiology of, 173
  - lesions of, 174
  - prognosis of, 179
  - prophylaxis of, 182
  - symptoms of, 176
  - treatment of, 180
- Acute follicular tonsillitis, diagnosis of, 47
  - symptoms of, 46
  - treatment of, 47
- Acute ileocolitis, course of lesions of, 175
  - description of, 173
  - diagnosis of, 178
  - etiology of, 173
  - lesions of, 174
  - prognosis of, 179
  - prophylaxis of, 182
  - symptoms of, 176
  - treatment of, 180
- Acute inflammatory diarrhea, course of lesions of, 175
  - description of, 173
  - diagnosis of, 178
  - etiology of, 173
  - lesions of, 174
  - prognosis of, 179
  - prophylaxis of, 182
  - symptoms of, 176

- Acute inflammatory diarrhea, treatment of, 180
- Acute intestinal indigestion, complications of, 170
- course of, 168
- diagnosis of, 169
- etiology of, 166
- prevention of, 170
- symptoms of, 168
- treatment of, 170
- Acute polioencephalitis, vomiting and, 95
- Acute toxic gastritis, symptoms and treatment of, 106
- Adenoidectomy, indications for, 51
- Adenoids, digestive system affected by, 50
- removal of, indications for, 51
- Agglutination, typhoid fever diagnosis by, 191
- Aglossia, 5
- Alcohol, accidental poisoning with, 311
- Alimentary allergy, clinical manifestations of, 318
- diagnosis of, 319
- incidence of, 316
- treatment of, 320
- Alimentary anemia, infancy and, 102
- Alkalies, accidental poisoning with, 310
- Allergy, alimentary, clinical manifestations of, 318
- diagnosis of, 319
- incidence of, 316
- treatment of, 320
- protein, clinical manifestations of, 318
- diagnosis of, 319
- incidence of, 317
- treatment of, 320
- Amebic dysentery, etiology of, 305
- lesions of, 306
- symptoms of, 307
- treatment of, 308
- Anemia, alimentary, infancy and, 102
- Angina, Vincent's, etiology and symptoms of, 41
- ulcerative stomatitis and, 27
- Angioma, lingual, 4
- Anorexia, asthenia causing, 220
- Anus, congenital abnormalities of, 287
- embryology of, 287
- fissure of, 293
- imperforate, 287
- prolapse of, 289
- Aphasia, typhoid fever and, 191
- Appendicitis, acute, anatomy of, 250
- affections mistaken for, 254
- Appendicitis, acute, diagnosis of, 253
- etiology of, 249
- pathology of, 250
- prognosis of, 255
- symptoms of, 251
- treatment of, 255
- Appendix, anatomy at birth, 250
- Appetite, deficient, 220
- perverted, 220
- Arsenic, accidental poisoning with, 311
- ulcerative stomatitis and, 29
- Artificially fed infant, constipation in, 223
- dyspepsia in, etiology of, 161
- physiological chemistry of, 162
- starch causing, 164
- symptoms of, 162
- treatment of, 163
- Ascaris lumbricoides, benign jaundice from, 328
- intestinal infestation with, 299
- Ascites, tuberculous peritonitis and, 280
- Aspidium, accidental poisoning with, 312
- Asthenia, anorexia caused by, 220
- Atony, gastric, 94
- Atresia, duodenal, diagnosis of, 236
- hypertrophic pyloric stenosis differentiated from, 120
- prognosis and treatment of, 237
- esophageal congenital, 72
- Atropin, accidental poisoning with, 312
- gastro-enterospasm of infancy and, 128
- hypertrophic pyloric stenosis and, 122
- Bacteria, stomach and, 91
- Balantidium coli, infestation with, 309
- Benign jaundice, round-worms causing, 328
- Bifid tongue, 5
- Bile ducts, absence of, 325
- Biliary passages, diseases of, gall-stones (cholelithiasis), 328
- Biliousness, hepatic insufficiency and, 327
- Blood, occult, stool and urine tested for, 343
- typhoid fever and, 189
- Bohn's epithelial pearls, 1
- Bothriocephalus latus, intestinal infestation with, 304
- Botulism, etiology, 315
- symptoms of, 316
- treatment of, 316
- Breast-fed infant, constipation in, 221
- dyspepsia in, diagnosis of, 160
- etiology of, 158

- Breast-fed infant, dyspepsia in, prognosis of, 161  
 — symptoms of, 159  
 — treatment of, 160  
 Breast feeding, diarrhea and, 149  
 Breasts, caking of, nursing and, 97  
 Bronchitis, typhoid fever and, 188
- Calcium, teeth and, 64  
 Camphor, accidental poisoning with, 312  
 Cancrum oris, 30  
 Carbohydrate, intestinal digestion of, 142  
 Caries, deciduous teeth and, 64  
 — permanent teeth and, 66  
 Catarrhal gastritis, acute, pathology of, 104  
 — symptoms of, 105  
 — treatment of, 105  
 Catarrhal jaundice, incidence of, 326  
 — treatment of, 327  
 Catarrhal pharyngitis, acute, treatment of, 39  
 Catarrhal stomatitis, etiology, 22  
 — symptoms and treatment of, 23  
 Caustic alkalies, accidental poisoning with, 310  
 Celiac disease, complications of, 213  
 — course of, 212  
 — diagnosis of, 213  
 — diet in, 215  
 — etiology of, 207  
 — nomenclature and history of, 206  
 — pathology and physiology of, 208  
 — prognosis of, 217  
 — symptoms of, 208  
 — treatment of, 214  
 Chenopodium, accidental poisoning with, 312  
 Cholecystitis, incidence of, 328  
 Cholelithiasis, incidence of, 328  
 Cholera infantum, 168  
 Chronic constipation, older children and, 226  
 Chronic intestinal indigestion, description of, 204  
 — treatment of, 205  
 Chronic gastric indigestion, treatment of, 107  
 Chronic ulcerative colitis, descriptive summary of, 198  
 — etiology of, 198  
 — lesions of, 199  
 — operative management of, 202  
 — symptoms of, 199  
 — treatment of, 201
- Cirrhosis, hepatic, etiology of, 330  
 Cleft-palate, disfiguration from, 6  
 — effect upon health, 7  
 — occurrence of, 6  
 — prognosis of, 10  
 — treatment of, 10  
 — when to operate on, 8  
 Colic, infantile, manifestations of, 99  
 — treatment of, 99  
 — types of, 98  
 Colitis, chronic ulcerative, descriptive summary of, 198  
 — etiology of, 198  
 — lesions of, 199  
 — operative management of, 202  
 — symptoms of, 199  
 — treatment of, 201  
 Colon, congenital dilatation and hypertrophy of, 239  
 — Hirschsprung's disease of, 239  
 — small, 245  
 Congenital dilatation and hypertrophy of colon (Hirschsprung's disease), 239  
 Congenital esophageal malformation, 72  
 Congenital megacolon, characteristics of, 239  
 Congenital microcolon, 245  
 Congenital pyloric stenosis, 113  
 Congenital volvulus, treatment of, 237  
 Constipation, artificially fed infant and, 223  
 — breast-fed infant and, 221  
 — chronic, diagnosis of, 229  
 — etiology of, 226  
 — older children and, 226  
 — symptoms of, 229  
 — treatment of, 230  
 — Hirschsprung's disease and, 241  
 — hypertrophic pyloric stenosis and, 116  
 — newborn and, 221  
 — treatment of, 222, 224  
 Cyclic vomiting, description of, 134  
 — diagnosis of, 136  
 — etiology of, 134  
 — prognosis of, 136  
 — symptoms of, 135  
 — treatment of, 137  
 Cyst, lingual, 6
- Deafness, typhoid fever and, 190  
 Deciduous teeth, abnormalities of, 63  
 — diseases of, 63  
 — eruption of, 60  
 — disturbances during, 62



- Deciduous teeth, eruption of, vomiting and, 94
- Dental hygiene, importance of, 69
- permanent teeth and, 71
  - prenatal care and, 70
  - preschool child and, 70
- Dentition, deciduous, 60
- abnormalities of, 63
  - permanent, 65
  - abnormalities of, 65
- De Silvestri's typhoid urine color reaction, 343
- Diarrhea, acute inflammatory, course of lesions of, 175
- description of, 173
  - diagnosis of, 178
  - etiology of, 173
  - lesions of, 174
  - prognosis of, 179
  - prophylaxis of, 182
  - symptoms of, 176
  - treatment of, 180
  - acute intestinal indigestion causing, 166
  - age incidence of, 152
  - classification of, 153
  - dyspepsia of artificially fed infants causing, 161
  - dyspepsia in breast-fed infants causing, 158
  - dyspepsia in premature infants causing, 153
  - etiology of, 153
  - feeding and, 150
  - general considerations of, 149
  - mortality from, 149
  - origin of, 167
  - starch indigestion causing, 164
  - tuberculous peritonitis and, 281
  - typhoid fever and, 187
- Diet, alimentary anemia and, 103
- celiac disease and, 215
  - chronic constipation in childhood and, 228, 230
  - cyclic vomiting and, 137
  - esophageal stricture and, 84
  - hypertrophic pyloric stenosis and, 119, 121
  - typhoid fever and, 192, 193
  - vomiting in infancy and, 93
- Digestive system, diseases of, abdominal examination in, 267
- abdominal tuberculosis, 276
  - abscess of gums, 13
  - accidental poisoning, 310-315
  - adenoid vegetations and, 50
  - actinomycosis of pharynx, 40
- Digestive system, diseases of, acute catarrhal gastritis, 104
- acute dysentery, 173
  - acute follicular tonsillitis, 46
  - acute ileocolitis, 173
  - acute inflammatory diarrhea, 173
  - acute intestinal indigestion, 166
  - acute pharyngitis, 39
  - acute tonsillitis, 40
  - acute toxic gastritis, 106
  - alimentary anemia in infancy, 102
  - amebic dysentery, 305
  - anal abnormalities and diseases, 287
  - anal fissure, 293
  - appendicitis, 249
  - atony of stomach, 94
  - biliary passage affections, 328
  - cancrum oris, 30
  - catarrhal stomatitis, 22
  - celiac disease, 206
  - chronic gastric indigestion, 107
  - chronic intestinal indigestion, 204
  - chronic ulcerative colitis, 198
  - colic in infancy, 98
  - congenital dilatation and hypertrophy of colon (Hirschsprung's disease), 239
  - congenital microcolon, 245
  - constipation, 221, 226
  - cyclic vomiting, 134
  - deficient appetite, 219
  - dental hygiene and, 69
  - diarrhea, 149, 166, 173
  - dilatation of, 108
  - disturbances in early infancy, 100
  - drug dosage for children, 342
  - dyspepsia of artificially fed infants, 161
  - dyspepsia in breast-fed infants, 158
  - dyspepsia in premature infants, 153
  - epidemic parotitis, 56
  - esophageal affections, 72, 74, 80
  - follicular stomatitis, 24
  - food poisoning, 315
  - foreign bodies in stomach, 111
  - frenal ulcer, 12
  - frozen milk and vomiting, 95
  - gangrenous stomatitis, 30
  - gastric hemorrhage, 108
  - gastric insufficiency, 94
  - gastro-enterospasm, 126
  - geographic tongue, 15
  - gingivitis, 13
  - glossitis, 16

- Digestive system, diseases of, hem-  
orrhoids, 293
- herpes of lip, 12
  - herpetic stomatitis, 24
  - Hirschsprung's disease, 239
  - hypertrophic pyloric stenosis, 113
  - hypertrophy of gums, 14
  - infantilism with fatty stools, 206
  - intestinal obstruction and mal-  
formation, 233
  - intestinal parasites, 295, 299, 303
  - intestines and, 139
  - intussusception, 255
  - liver affections, 324
  - maculofibrinous stomatitis, 24
  - malformations and tumors, of  
stomach, 113
  - mechanical vomiting, 92
  - Meckel's diverticulum and, 245
  - mercurial stomatitis, 36
  - Mikulicz's disease, 55
  - necrosis of jaw, 16
  - noma, 30
  - non-epidemic parotitis, 56
  - nursing and, 97
  - pancreas affections, 331
  - paratyphoid fever, 195
  - peptic ulcer of duodenum, 145
  - periostitis of gums, 13
  - peritonitis, 261, 276
  - peritonsillar abscess, 42
  - poisoning, 310
  - proctitis, 291
  - pylorospasm, 126
  - quinsy, 42
  - rectal abnormalities and diseases,  
287
  - rectal fissure, 293
  - rectal polypus, 294
  - retropharyngeal abscess, 43
  - retropharyngeal lymphadenitis, 43
  - Riga's disease of frenum, 12
  - rôle of lymphoid disease in pharynx  
and fauces, 48
  - rumination, 130
  - salivary gland affections, 52
  - salivary stones, 58
  - sarcoma of jaw, 17
  - scarlatinal tonsillitis, 41
  - scurvy of gums, 14
  - simple regurgitation, 92
  - starch causing digestive injury, 164
  - starvation fever in newborn, 101
  - stomach disturbances, 86, 104
  - stomatitis, 19
  - sublingual inflammation, 58
  - submaxillary inflammation, 58
- Digestive system, diseases of, syphilis af-  
fecting salivary glands, 59
- syphilis of mouth, 17
  - technical methods in pediatric prac-  
tice, 335
  - teeth and, 60, 65
  - thrush, 19
  - tongue-tie, 2
  - tonsillar diphtheria, 40
  - toxic vomiting, 95
  - tumors and deformities of tongue,  
4
  - tumors of jaw, 16
  - typhoid fever, 184
  - ulceration of hard palate, 35
  - ulcerative stomatitis, 26
  - ulcer of stomach, 109
  - Vincent's angina, 41
  - visceroptosis and, 270
  - vomiting in infancy, 92
  - vomiting of central origin, 95
  - vomiting of intestinal influenza, 95
  - worms, 295, 299, 304, 309
  - x-ray diagnosis of, 335
  - gastric, physiology of, 89
  - intestinal, 141
- Dilatation, gastric, 108
- Diphtheria, tonsillar, 40
- Diverticulum, Meckel's, disturbances of,  
245
- Dosage, drug, formulæ for, 342
- Dover's powder, acute intestinal indi-  
gestion and, 172
- Drug dosage, formulæ for, 342
- Duodenum, atresia of, hypertrophic  
pyloric stenosis differentiated  
from, 120
- malformation of, 236
  - obstruction of, 236
  - peptic ulcers of, 145
  - physiology of, 140
- Dysentery, acute, celiac disease differen-  
tiated from, 214
- course of lesions of, 175
  - description of, 173
  - diagnosis of, 178
  - etiology of, 173
  - lesions of, 174
  - prognosis of, 179
  - prophylaxis of, 182
  - symptoms of, 176
  - treatment of, 180
  - typhoid fever differentiated from,  
192
  - amebic, etiology of, 305
  - lesions of, 306
  - symptoms of, 307

- Dysentery, amebic, treatment of, 308
- Dyspepsia, artificially fed infant, etiology of, 161
  - physiological chemistry of, 162
  - starch causing, 164
  - symptoms of, 162
  - treatment of, 163
- breast-fed infant, diagnosis of, 160
  - etiology of, 158
  - prognosis of, 161
  - symptoms of, 159
  - treatment of, 160
- premature infant, diarrhea caused by, 153
- premature infant, treatment of, 156

Encephalitis, epidemic, vomiting and, 95

Entamœba histolytica, intestinal infestation with, 305

Epidemic encephalitis, vomiting and, 95

Epidemic salivary gland infection, 56

Epistaxis, typhoid fever and, 187

Epithelial pearls, Bohn's, 1

Epulis, 17

Esophagus, anatomy of, 75

— congenital atresia of, 72

— congenital malformation of, 72

— congenital narrowing of, 74

— diseases of, 74

— foreign bodies in, 76

— physiology of, 75

— safety pins in, 76

— spasm of, 80

— stricture of, course of, 81

— diagnosis of, 83

— pathology of, 80

— symptoms of, 81

— treatment of, 83

Essential purpura, 273

Exhaustion, heat, infancy and, 102

Fat, intestinal digestion of, 141

Fatty degeneration, liver and, 330

Fatty stools, infantilism with, 206

Fauces, lymphoid disease and, 48

Fecal impaction, intestinal obstruction caused by, 239

Feeding, sour milk, 338

— thick, 337

— tube, 338

Fetal peritonitis, 261

Fever, paratyphoid, bacteriology of, 195

— complications of, 197

— diagnosis of, 198

— etiology of, 195

Fever, paratyphoid, general considerations of, 195

— lesions of, 196

— prognosis of, 198

— symptoms of, 196

— treatment of, 198

— pollen, susceptibility and, 317

— starvation, newborn and, 101

— thermic, infancy and, 102

— typhoid, age incidence of, 184

— complications of, 190

— course of, 190

— descriptive summary of, 184

— De Silvestri's color reaction in urine in, 343

— diagnosis of, 191

— etiology of, 185

— incidence of, 184

— incubation period of, 187

— miliary tuberculosis and, 191

— pathology of, 185

— plateau and stepladder types of, temperature in, 187

— pneumonia and, 191

— prognosis of, 194

— prophylaxis of, 194

— relapse in, 190

— symptoms of, 186

— treatment of, 192

— vaccine therapy of, 193

— Widal reaction in, 342

Fissure, anal, 293

— rectal, 293

Follicular tonsillitis, acute, diagnosis of, 47

— symptoms of, 46

— treatment of, 47

Follicular stomatitis, 24

Food, regurgitation of, 130

— vomiting in infancy and, 93

Food poisoning, alimentary allergy, 316

— botulism, 315

— milk-sickness, 320

Formaldehyd, accidental poisoning with, 313

Frenum, labial, Riga's disease of, 12

— simple ulcer of, 12

— lingual, tongue-swallowing and, 4

— tongue-tie and, 2

Frozen milk, vomiting in infancy and, 95

Furunculosis, typhoid fever and, 190

Gall-stones, incidence of, 328

Gangrenous stomatitis, diagnosis of, 33

— etiology, 31

— lesions of, 32

- Gangrenous stomatitis, nomenclature of, 30
- prophylaxis of, 35
  - symptoms of, 32
  - treatment of, 34
- Gastric digestion, physiology of, 89
- Gastric hemorrhage, 108
- Gastric indigestion, chronic, treatment of, 107
- Gastric insufficiency, 94
- Gastric ulcer, causes of, 110
- occurrence of, 109
  - pathology of, 110
  - symptoms of, 111
- Gastritis, acute catarrhal, pathology of, 104
- symptoms of, 105
  - acute toxic, symptoms and treatment of, 106
- Gastro-enterospasm, clinical description of, 126
- diagnosis and differentiation of, 128
  - etiology of, 126
  - prognosis of, 129
  - symptoms of, 127
  - treatment of, 127
- Gavage, technic of, 338
- Geographic tongue, 15
- Giardia intestinalis, infestation with, 309
- Gingivitis, 13
- Glands, lacrimal, Mikulicz's disease affecting, 55
- salivary, abnormal development of, 53
  - absence of, 54
  - anatomical and functional considerations of, 52
  - cyst of, 54
  - disturbances of secretion of, 53
  - epidemic infection of, 56
  - lymphatic enlargement affecting, 59
  - Mikulicz's disease of, 55
  - non-epidemic parotitis, 56
  - stones obstructing ducts of, 58
  - syphilis affecting, 59
  - sublingual, inflammation of, 58
  - submaxillary, inflammation of, 58
- Glossitis, 16, 17
- Gonorrheal proctitis, treatment of, 292
- Grenouillette, definition of, 54
- Gums, abscess of, 13
- diseases of, 13
  - hypertrophy of, 14
  - periostitis of, 13
  - scurvy of, 14
- Hard palate, ulceration of, 35
- Harelip, disfiguration from, 6
- effect upon health, 7
  - occurrence of, 6
  - prognosis of, 10
  - treatment of, 10
  - when to operate upon, 8
- Hay-fever, susceptibility and, 317
- Heat exhaustion, infancy and, 102
- Hemangioma, parotid, 53
- Hemophilia, purpura hæmorrhagica differentiated from, 274
- Hemorrhage, gastric, 108
- typhoid fever complicated by, 190
- Hemorrhoids, treatment of, 294
- Henoch's purpura, diagnosis of, 274
- intussusception differentiated from, 260
  - symptoms of, 273
- Hepatic insufficiency, biliousness and, 327
- Hepatitis, infectious, etiology of, 325
- Heredity, sensitization and, 317
- Herpes labialis, 12
- Herpetic stomatitis, etiology of, 24
- symptoms of, 25
  - treatment of, 26
- Hirschsprung's disease, anatomy of, 240
- celiac disease differentiated from, 213
  - characteristics of, 239
  - course of, 244
  - diagnosis of, 242
  - etiology of, 240
  - pathology of, 240
  - prognosis of, 244
  - symptoms of, 241
  - treatment of, 243
- Hookworm, intestinal infestation with, 303
- Hunger, stomach and effects of, 91
- Hutchinson's teeth, 69
- Hydatid disease, liver and, 330
- Hydrocephalus, vomiting and, 95
- Hygiene, dental, importance of, 69
- permanent teeth and, 71
  - prenatal care and, 70
  - preschool child and, 70
- Hyperperistalsis, hypertrophic pyloric stenosis and, 116
- Hypertrophic pyloric stenosis, diagnosis and differentiation of, 119
- etiology of, 114
  - incidence of, 114
  - nomenclature and history of, 113
  - pathology and physiology of, 115
  - prognosis of, 117, 124
  - symptoms of, 113, 115
  - treatment of, 121



- Hypertrophic pyloric stenosis, treatment of, surgical, 123
- Hypertrophy, gingival, 14
- Hypodermoclysis, technic of, 340
- Ileocolitis, acute, course of lesions of, 175
- description of, 173
  - diagnosis of, 178
  - etiology of, 173
  - intussusception differentiated from, 260
  - lesions of, 174
  - prognosis of, 179
  - prophylaxis of, 182
  - symptoms of, 176
  - treatment of, 180
- Impaction, fecal, intestinal obstruction caused by, 239
- Imperforate anus, 287
- Indigestion, acute intestinal, complications of, 170
- course of, 168
  - diagnosis of, 169
  - etiology of, 166
  - prevention of, 170
  - symptoms of, 168
  - treatment of, 170
- chronic intestinal, description of, 204
  - treatment of, 205
  - chronic gastric, treatment of, 107
- Infancy, alimentary anemia in, 102
- colic in, manifestations of, 99
  - treatment of, 99
  - types of, 98
  - early, digestive disturbances in, 100
  - gastro-enterospasm of, 126
  - heat exhaustion in, 102
  - hypertrophic pyloric stenosis of, 113
  - nursing and alimentary disturbances in, 97
  - paratyphoid fever in, symptoms of, 196
  - pylorospasm of, 126
  - rumination in, symptoms of, 131
  - stools in, 142
  - thermic fever in, 102
  - vomiting in, 92
  - causes inherent in food, 93
  - central origin of, 95
  - eruption of teeth and, 94
  - frozen milk and, 95
  - intestinal influenza and, 95
  - mechanical vomiting, 92
  - simple regurgitation, 92
  - toxic, 95
- Infancy, vomiting in, treatment of, 96
- Infant, artificially fed, constipation in, 223
- breast-fed, constipation in, 221
  - nursing, cleft-palate and harelip and, 9
  - difficulties encountered while, 97
- Infantile peritonitis, 261
- Infantilism, fatty stools and, 206
- Infectious hepatitis, etiology of, 325
- Infectious purpura, 273
- Inflammatory diarrhea, acute, course of lesions of, 175
- description of, 173
  - diagnosis of, 178
  - etiology of, 173
  - lesions of, 174
  - prognosis of, 179
  - prophylaxis of, 182
  - symptoms of, 176
  - treatment of, 180
- Influenza, intestinal, vomiting in, 95
- Injection, intraperitoneal, technic of, 341
- Injury, permanent teeth and, 68
- Inspection, abdominal, 267
- Insufficiency, gastric, 94
- hepatic, biliousness and, 327
- Intestinal indigestion, acute, complications of, 170
- course of, 168
  - diagnosis of, 169
  - etiology of, 166
  - prevention of, 170
  - symptoms of, 168
  - treatment of, 170
  - chronic, description of, 204
  - treatment of, 205
- Intestinal influenza, vomiting in, 95
- Intestinal parasites, general considerations of, 295
- varieties of, 296, 299, 303, 305, 309
- Intestines, abnormalities of, 140
- anatomy of, 139
  - bacteria in, 144
  - carbohydrate digestion in, 142
  - congenital obstruction of, 233
  - digestion in, 141
  - diseases of, acute dysentery, 173
  - acute ileocolitis, 173
  - acute inflammatory diarrhea, 173
  - acute intestinal indigestion, 166
  - acute surgical, 249
  - amebic dysentery, 305
  - appendicitis, 249
  - celiac disease, 206
  - chronic intestinal indigestion, 204

Intestines, diseases of, chronic ulcerative colitis, 198

- congenital dilatation and hypertrophy of colon (Hirschsprung's disease), 239
- constipation, 221, 226
- diarrhea, 149
- dyspepsia in artificially fed infants, 161
- dyspepsia in breast-fed infants, 158
- dyspepsia in premature infants causing diarrhea, 153
- Hirschsprung's disease, 239
- infantilism with fatty stools, 206
- intussusception, 255
- malformation and obstruction, 233
- Meckel's diverticulum and, 245
- parasitic, 295
- paratyphoid fever, 195
- peptic duodenal ulcer, 145
- peritonitis, 261, 276
- protozoa causing, 296, 299, 303
- starch causing digestive injury, 164
- tuberculosis, 276, 279
- typhoid fever, 184
- visceroptosis, 270
- worms, 295, 299, 304, 309
- duodenum, physiology of, 140
- emptying time of, 139
- extrinsic obstruction of, 236
- fat digestion in, 141
- length and position of, 139
- malformation of, congenital micro-colon, 245
- diagnosis of, 236
- etiology of, 233
- Hirschsprung's disease and, 239
- symptoms of, 234
- obstruction of, diagnosis of, 236
- fecal impaction causing, 239
- intestinal stone causing, 239
- symptoms of, 234
- peculiarities of, 140
- physiology of, 139
- protein digestion in, 141
- starch digestion in, 142
- stenosis of, symptoms of, 235
- stools in infancy, 142
- sugar digestion in, 142

Intraperitoneal injection, technic of, 341

Intussusception, anatomy and pathology of, 256

- course of, 258
- description of, 255
- diagnosis of, 260
- etiology of, 256
- symptoms of, 257

Intussusception, treatment of, 260

Invagination, intestinal, 255

Jaundice, benign, round-worms causing, 328

- catarrhal, incidence of, 326
- treatment of, 327

Jaw, necrosis of, 16

- sarcoma of, 17
- tumor of, 16

Kidneys, typhoid pathology of, 191, 192

Lacrimal gland, Mikulicz's disease affecting, 55

Lambliia intestinalis, infestation with, 309

Latent peritonitis, 261

Lavage, gastric, methods of, 339

Lead, accidental poisoning with, 313

Lip, herpes of, 12

Liver, diseases of, benign jaundice from round-worms, 328

- catarrhal jaundice, 326

- cirrhosis, 330

- fatty degeneration, 330

- hepatic insufficiency, 327

- hydatid disease, 330

- infectious hepatitis, 325

- early life disturbances of, 325

- physiology of, 324

- tumors of, 325

- typhoid pathology and, 186

Lobar pneumonia, acute appendicitis differentiated from, 253

Lymphadenitis, retropharyngeal, 43

Lymphangioma, salivary gland, 54

Macroglossia, 5

Maculofibrinous stomatitis, 24

Malocclusion, 68

Mechanical vomiting, 92

Meckel's diverticulum, disturbances of, 245

Megacolon, congenital, 239

Mehlnährschaden, 142

Melena, bleeding in newborn or, 109

- peptic ulcer of duodenum and, 146

Meningitis, syphilitic, vomiting and, 95

- tuberculous, vomiting and, 95

Mercurial stomatitis, 36

Mercury, accidental poisoning with, 313

Meyer's solution, occult blood in stool and urine and, 343

- Microcolon, congenital, 245  
 Microglossia, 5  
 Mikulicz's disease, glands affected by, 55  
 Miliary tuberculosis, typhoid fever and, 191  
 Milk, diarrhea and, 151  
 — frozen, vomiting in infancy and, 95  
 Milk-sickness, animals affected by, 321  
 — etiology of, 321  
 — incidence of, 320  
 — symptoms of, 322  
 — treatment of, 322  
 Morbus maculosus, 273  
 Mouth, characteristics at birth, 1  
 — diseases of, abscess of gums, 13  
 — — cancrum oris, 30  
 — — catarrhal stomatitis, 22  
 — — follicular stomatitis, 24  
 — — frenal ulcer, 12  
 — — gangrenous stomatitis, 30  
 — — geographic tongue, 15  
 — — gingivitis, 13  
 — — glossitis, 16  
 — — herpes of lips, 12  
 — — herpetic stomatitis, 24  
 — — hypertrophy of gums, 14  
 — — maculofibrinous stomatitis, 24  
 — — mercurial stomatitis, 36  
 — — necrosis of jaw, 16  
 — — noma, 30  
 — — periostitis of gums, 13  
 — — Riga's disease, 12  
 — — sarcoma of jaw, 17  
 — — scurvy of gums, 14  
 — — stomatitis, 19  
 — — syphilis, 17  
 — — thrush, 19  
 — — tumor of, 16  
 — — ulceration of hard palate, 35  
 — — ulcerative stomatitis, 26  
 — inflammation of, 19  
 — malformations of structure of, 2  
 — — cleft-palate and harelip, 6  
 — — cyst, 6  
 — — tongue-swallowing and, 4  
 — — tongue-tie, 2  
 — — tumors and deformities of tongue, 4  
 — microorganisms at birth in, 2  
 — sucking mechanism of, 2  
 — syphilis of, 17  
 Mumps, glands affected by, 56  
 Narrowing, esophageal, congenital, 74  
 Necator americana, intestinal infestation with, 303  
 Necrosis, maxillary, 16  
 Necrotic tonsillitis, Vincent's, 41  
 Neo-arsphenamin, ulcerative stomatitis and, 30  
 Newborn, constipation in, 221  
 — starvation fever in, 101  
 — stomach of, capacity of, 87  
 — stools of, 142  
 — volvulus in, 237  
 Noma, 30  
 Nursing, infant, cleft-palate and harelip and, 9  
 — — difficulties encountered in, 97  
 — typhoid fever and, 192  
 Obstruction, intestinal, 233  
 Occult blood, stool and urine tested for, 343  
 Oidium albicans, thrush and, 20  
 Omphalomesenteric duct, Meckel's diverticulum and, 245  
 Opium, accidental poisoning with, 313  
 — acute intestinal indigestion and, 172  
 — typhoid fever and, 193  
 Osteomyelitis, typhoid, 191  
 Otitis media, typhoid fever and, 190  
 Oxyuris vermicularis, intestinal infestation with, 296  
 Pain, abdominal, causes of obscure type of, 269  
 Palate, hard, ulceration of, 35  
 — soft, actinomycosis of, 40  
 Palpation, abdominal, 268  
 Pancreas, diseases of, 331  
 Papilloma, lingual, 5  
 Parasites, intestinal, general considerations of, 295  
 — — varieties of, 296, 299, 303, 305, 309  
 Paratyphoid fever, bacteriology of, 195  
 — complications of, 197  
 — diagnosis of, 198  
 — etiology of, 195  
 — general considerations of, 195  
 — lesions of, 196  
 — prognosis of, 198  
 — symptoms of, 196  
 — treatment of, 198  
 Paregoric, acute intestinal indigestion and, 172  
 Parotid glands, abnormal development of, 53  
 — anatomical and functional considerations of, 52

- Parotid glands, disturbances of secretion of, 53  
 — epidemic infection of, 56  
 — hemangioma of, 53  
 — lymphangioma of, 54  
 — lymphatic enlargement affecting, 59  
 — mixed tumors of, 54  
 — non-epidemic infection of, 56  
 Peptic ulcer, chronic, 110  
 — duodenum and, 145  
 Percussion, abdominal, 269  
 Perforation, Meckel's diverticulum and, 247  
 Periostitis, gingival, 13  
 Peristalsis, hypertrophic pyloric stenosis causing increase of, 116  
 Peritonitis, fetal, 261  
 — infantile, 261  
 — latent, 261  
 — pneumococcic, 264  
 — static, 262  
 — streptococcic, 263  
 — tuberculous, celiac disease differentiated from, 214  
 — — complications of, 283  
 — — diagnosis of, 282  
 — — lesions of, 280  
 — — prognosis of, 284  
 — — symptoms of, 280  
 — — treatment of, 282  
 — typhoid, perforation and, 186, 190  
 Peritonsillar abscess, symptoms and treatment of, 42  
 Permanent teeth, abnormalities of, 65  
 — caries of, 66  
 — eruption of, 65  
 — hygiene for, 71  
 — malocclusion of, 68  
 — syphilis of, 69  
 — trauma of, 68  
 Perverted appetite, 220  
 Peyer's patches, typhoid pathology and, 186  
 Pharyngitis, acute catarrhal, treatment of, 39  
 — anatomical characteristics of, 37  
 — congenital obstruction of, 37  
 Pharynx, diseases of, actinomycosis, 40  
 — acute follicular tonsillitis, 46  
 — — acute pharyngitis, 39  
 — — acute tonsillitis, 40  
 — — peritonsillar abscess, 42  
 — — quinsy, 42  
 — — retropharyngeal abscess, 43  
 — — retropharyngeal lymphadenitis, 43  
 — — rôle of lymphoid disease in, 48  
 — — scarlatinal tonsillitis, 41  
 Pharynx, diseases of, Vincent's angina, 41  
 — foreign bodies in, 37  
 — lymphoid disease and, 48  
 Phenol, accidental poisoning with, 311  
 Phosphorus, accidental poisoning with, 314  
 — teeth and, 64  
 Pica, 220  
 Pink disease, teeth and, 64  
 Pin-worm, intestinal infestation with, 296  
 Pneumococcic peritonitis, 264  
 Pneumonia, lobar, acute appendicitis differentiated from, 253  
 — typhoid fever complicated by, 190, 191  
 Poisoning, accidental, acids, 311  
 — — alcohol, 311  
 — — alkalies, 310  
 — — arsenic, 311  
 — — aspidium, 312  
 — — atropin, 312  
 — — camphor, 312  
 — — chenopodium, 312  
 — — formaldehyd, 313  
 — — lead, 313  
 — — mercury, 313  
 — — opium, 313  
 — — phosphorus, 314  
 — — santonin, 314  
 — — silver nitrate, 314  
 — — turpentine, 314  
 — food, alimentary allergy, 316  
 — — botulism, 315  
 — — milk-sickness, 320  
 Pollen fever, susceptibility and, 317  
 Polypus, rectal, 294  
 Polioencephalitis, acute, vomiting and, 95  
 Posture, visceral ptosis and, 270  
 Premature infant, dyspepsia in, diarrhea caused by, 153  
 — — treatment of, 156  
 Prenatal care, dental hygiene and, 70  
 Preschool child, dental hygiene and, 70  
 Proctitis, etiology of, 291  
 — gonorrheal, treatment of, 292  
 Proctoclysis, technic of, 340  
 Protein, intestinal digestion of, 141  
 Protein allergy, 319  
 Protein sensitization, susceptibility and, 317  
 Ptosis, visceral, 270  
 Ptyalism, causes of, 53  
 Purpura, abdominal, diagnosis of, 274  
 — — symptoms of, 273  
 — — treatment of, 274



- Purpura, essential, 273  
 — Henoch's, diagnosis of, 274  
 — — intussusception differentiated from, 260  
 — — symptoms of, 273  
 — — treatment of, 274  
 — infectious, 273  
 Purpura hæmorrhagica, hemophilia differentiated from, 274  
 — symptoms of, 273  
 — treatment of, 274  
 Pyelitis, typhoid, 191, 192  
 Pyloric stenosis, hypertrophic, diagnosis and differentiation of, 119  
 — — etiology of, 114  
 — — incidence of, 114  
 — — nomenclature and history of, 113  
 — — pathology and physiology of, 115  
 — — prognosis of, 117, 124  
 — — symptoms of, 113, 115  
 — — treatment of, 121  
 — — — surgical, 123  
 Pylorospasm, clinical picture of, 126  
 — diagnosis and differentiation of, 128  
 — etiology of, 126  
 — prognosis of, 129  
 — symptoms of, 127  
 — treatment of, 127  
 Pylorospasmus, 113
- Quinsy, symptoms and treatment of, 42
- Ranula, definition of, 54  
 Rectum, congenital abnormalities of, 287  
 — embryology of, 287  
 — fissure of, 293  
 — polypus of, 294  
 — prolapse of, 289  
 Recurrent vomiting, 134  
 Regurgitation, simple, infancy and, 92  
 Retropharyngeal abscess, diagnosis, 44  
 — symptoms of, 43  
 — treatment of, 45  
 Retropharyngeal lymphadenitis, 43  
 Rhagades, nursing rendered difficult by, 98  
 Rhinitis, syphilitic, nursing hindered by, 98  
 Rickets, celiac disease differentiated from, 214  
 — teeth and, 64  
 Riga's disease, 12  
 Rose spots, typhoid fever and, 188  
 Round-worms, benign jaundice from, 328  
 — intestinal infestation with, 299
- Rumination, course of, 132  
 — description of, 130  
 — diagnosis of, 132  
 — etiology of, 130  
 — mechanism of, 130  
 — symptoms of, 131  
 — treatment of, 132
- Salivary glands, abnormal development of, 53  
 — absence of, 54  
 — anatomical and functional considerations of, 52  
 — cysts of, 54  
 — diseases of, epidemic parotitis, 56  
 — — parotitis (non-epidemic), 56  
 — — sublingual inflammation, 58  
 — — submaxillary inflammation, 58  
 — disturbances of secretion of, 53  
 — lymphatic enlargement affecting, 59  
 — Mikulicz's disease of, 55  
 — stones obstructing ducts of, 58  
 — syphilis affecting, 59  
 Salivation, causes of, 53  
 Santonin, accidental poisoning with, 314  
 Sarcoma, salivary gland, 17, 55  
 Scarlatinal tonsillitis, 41  
 Scurvy, gingival, 14  
 Seat-worm, intestinal infestation with, 296  
 Septic sore-throat, ulcerative stomatitis and, 27  
 Serum, dysentery, 182  
 Silver nitrate, accidental poisoning with, 314  
 Simple regurgitation, 92  
 Sisto's sign, colic in infancy and, 100  
 — congenital syphilis and, 129  
 Snuffles, nursing hindered by, 98  
 Soft palate, actinomycosis of, 40  
 Sore-throat, septic, ulcerative stomatitis and, 27  
 Sour milk feeding, 338  
 Spasm, esophageal, 80  
 Spleen, typhoid pathology and, 186  
 Starch, digestive injury from, 164  
 — intestinal digestion of, 142  
 Starvation fever, newborn and, 101  
 Static peritonitis, 262  
 Stenosis, hypertrophic pyloric, diagnosis and differentiation of, 119  
 — — etiology of, 114  
 — — nomenclature and history of, 113  
 — — incidence of, 114  
 — — pathology and physiology of, 115

- Stenosis, hypertrophic pyloric, prog-  
     nosis of, 117, 124
- symptoms of, 113, 115
- treatment of, 121
- — surgical, 123
- intestinal, diagnosis of, 236
- symptoms of, 235
- Stomach, anatomy of, 86
- atony of, 94
- bacteria in, 91
- capacity in infancy, 87
- digestion in, physiology of, 89
- digestive function in infancy, 88
- diseases and disturbances of, acute  
     gastritis, 104
- acute toxic gastritis, 106
- atony of stomach, 94
- chronic gastric indigestion, 107
- cyclic vomiting, 134
- dilatation, 108
- foreign bodies, 111
- frozen milk causing, 95
- gastric insufficiency, 94
- gastro-enterospasm, 126
- hemorrhage, 108
- hypertrophic pyloric stenosis, 113
- intestinal influenza, 95
- malformations, 113
- mechanical vomiting, 92
- rumination, 130
- simple regurgitation, 92
- toxic vomiting, 95
- tumors, 113
- ulcer, 109
- vomiting in infancy, 92
- vomiting of central origin, 95
- emptying time in infancy, 87
- foreign bodies in, treatment of,  
     112
- variety of, 111
- hunger affecting, 91
- lavage of, methods of, 339
- malformations of, 113
- physiology of, 86, 89
- tumors of, 113
- ulcer of, causes of, 110
- — occurrence of, 109
- — pathology of, 110
- — symptoms of, 111
- Stomatitis, catarrhal, 22
- etiology of, 19
- follicular, 24
- gangrenous, diagnosis of, 33
- — etiology of, 31
- — lesions of, 32
- — nomenclature of, 30
- — prophylaxis of, 35
- Stomatitis, gangrenous, symptoms of, 32
- — treatment of, 34
- herpetic, etiology of, 24
- — symptoms of, 25
- — treatment of, 26
- maculofibrinous, 24
- mercurial, 36
- ulcerative, 26
- — diagnosis of, 29
- — etiology of, 27
- — lesions of, 27
- — prophylaxis of, 29
- — symptoms of, 28
- — treatment of, 29
- — Vincent's angina and, 27
- Stone, intestinal, obstruction caused by,  
     239
- salivary, 58
- Stool, blood in, 144, 343
- fat in, 143
- fatty, infantilism with, 206
- infantile, breast feeding and, 143
- — newborn, 142
- occult blood in, test for, 343
- salt content of, 144
- Streptococcic peritonitis, 263
- Stricture, esophageal, course of, 81
- — diagnosis of, 83
- — pathology of, 80
- — symptoms of, 81
- — treatment of, 83
- Sublingual glands, anatomical and func-  
     tional considerations of, 52
- disturbances of secretion of, 53
- inflammation of, 58
- Submaxillary glands, anatomical and  
     functional considerations of, 52
- disturbances of secretion of, 53
- inflammation of, 58
- Sucking, mechanism of, 2
- Sucking pad, function of, 1
- Sulpharsphenamin, ulcerative stomatitis  
     and, 30
- Sugar, intestinal digestion of, 142
- Susceptibility, protein allergy, 317
- Syphilis, lingual, 17
- oral, 17
- permanent teeth and, 69
- salivary glands affected by, 59
- Syphilitic meningitis, vomiting and, 95
- Syphilitic rhinitis, nursing hindered by,  
     95
- Tænia saginata, intestinal infestation  
     with, 304
- Tænia solium, intestinal infestation  
     with, 304

- Tapeworm, intestinal infestation with, 304
- Teeth, deciduous, abnormalities of, 63
- diseases of, 63
- disturbances during eruption of, 62
- eruption of, 60
- loss of, 61
- eruption of, vomiting in infancy and, 94
- foods for, 70
- Hutchinson's, 69
- hygiene of, 69
- permanent, abnormalities of, 65
- caries of, 66
- eruption of, 65
- hygiene for, 71
- malocclusion of, 68
- syphilis of, 69
- trauma of, 68
- Thread-worm, intestinal infestation with, 304
- Thermic fever, infancy and, 102
- Thick feeding, 337
- Thrombosis, typhoid pathology and, 186
- Thrush, diagnosis of, 21
- etiology of, 19
- lesions of, 20
- prognosis of, 22
- symptoms of, 21
- treatment of, 22
- Tongue, angioma of, 4
- bifid, 5
- congenital hypertrophy of, 5
- cysts of, 6
- deformities of, 4
- double, 5
- geographic, 15
- inflammation of, 16
- papilloma of, 5
- syphilis of, 17
- tumors of, 4
- Tongue-swallowing, 4
- Tongue-tie, anatomical considerations of, 2
- operative management of, 4
- treatment of, 3
- Tonsillar diphtheria, 40
- Tonsillectomy, indications for, 50, 51
- Tonsillitis, acute follicular, diagnosis of, 47
- symptoms of, 46
- treatment of, 47
- scarlatinal, 41
- Vincènt's ulcerative, 41
- Tonsils, anatomy of, lymphoid disease affecting, 48
- Tonsils, diseases of, acute follicular tonsillitis, 46
- acute tonsillitis, 40
- peritonsillar abscess, 42
- scarlatinal tonsillitis, 41
- tonsillar diphtheria, 40
- Vincent's angina, 41
- lymphoid disease causing anatomical changes in, 48
- removal of, indications for, 50, 51
- Toxic gastritis, acute, symptoms and treatment of, 106
- Toxic vomiting, infancy and, 95
- Trauma, permanent teeth and, 68
- Trench mouth, ulcerative stomatitis and, 27
- Trichocephalus dispar, intestinal infestation with, 304
- Trichuris trichiura, intestinal infestation with, 304
- Tube feeding, 338
- Tuberculin test, tuberculous peritonitis and, 283
- Tuberculosis, abdominal, complications of, 283
- diagnosis of, 282
- etiology of, 277
- general survey of, 276
- intestinal lesions of, 279
- mesenteric lesions of, 280
- occurrence of, 277
- pathogenesis of, 277
- peritoneal lesions of, 280
- prognosis of, 284
- symptoms of, 280
- treatment of, 284
- miliary, typhoid fever and, 192
- Tuberculous meningitis, vomiting and, 95
- Tuberculous peritonitis, celiac disease differentiated from, 214
- complications of, 283
- diagnosis of, 282
- lesions of, 280
- prognosis of, 284
- symptoms of, 280
- treatment of, 284
- Tumors, hypertrophic pyloric spasm and, 116
- lingual, 4
- liver, 325
- maxillary, 16
- parotid, 53, 54
- stomach diseases and, 113
- Turpentine, accidental poisoning with, 314
- Typhoid fever, age incidence of, 184

- Typhoid fever, complications of, 190
  - course of, 190
  - descriptive summary of, 184
  - De Silvestri's color reaction in urine in, 343
  - diagnosis of, 191
  - etiology of, 185
  - incidence of, 184
  - incubation period of, 187
  - miliary tuberculosis and, 191
  - pathology of, 185
  - plateau and stepladder types of temperature in, 187
  - pneumonia and, 191
  - prognosis of, 194
  - prophylaxis of, 194
  - relapse in, 190
  - symptoms of, 186
  - treatment of, 192
  - vaccine therapy in, 193
  - Widal reaction in, 342
- Ulcer, frenal, simple, 12
  - gastric, causes of, 110
  - occurrence of, 109
  - pathology of, 110
  - symptoms of, 111
  - hard palate, 35
  - Meckel's diverticulum and, 246
- Ulcerative colitis, chronic, descriptive summary of, 198
  - etiology of, 198
  - lesions of, 199
  - operative management of, 202
  - symptoms of, 199
  - treatment of, 201
- Ulcerative stomatitis, 26
  - diagnosis of, 29
  - etiology of, 27
  - lesions of, 27
  - prophylaxis of, 29
  - symptoms of, 28
  - treatment of, 29
  - Vincent's angina and, 27
- Ulcerative tonsillitis, Vincent's, 41
- Urine, occult blood in, test for, 343
  - typhoid, De Silvestri's color reaction in, 343
  - typhoid fever and, 188
- Urotropin, typhoid fever and, 193
- Uvula, elongated, 38
  - syphilis affecting, 18
- Vaccine, colitis and, 202
  - dysentery, 183
  - paratyphoid prophylaxis and, 198
  - typhoid, 193
- Vagotonia, 126
- Vegetations, adenoid, digestive system affected by, 50
- Vincent's angina, etiology symptoms and treatment of, 41
  - ulcerative stomatitis and, 27
- Viscerotoposis, gastro-intestinal disturbances due to, 270
  - manifestations of, 271
  - treatment of, 272
- Vitamins, teeth and, 64
- Volvulus, congenital, treatment of, 237
  - Meckel's diverticulum and, 246
- Vomiting, cyclic, description of, 134
  - diagnosis of, 136
  - etiology of, 134
  - prognosis of, 136
  - symptoms of, 135
  - treatment of, 137
  - hemorrhage and, 108
  - hypertrophic pyloric stenosis and, 115, 120
  - infant and, 92
  - causes inherent in food, 93
  - eruption of teeth and, 94
  - frozen milk and, 95
  - intestinal influenza and, 95
  - mechanical vomiting, 92
  - simple regurgitation, 92
  - toxic vomiting, 95
  - treatment of, 96
  - vomiting of central origin, 95
- Water, administration of, methods of, 339
- Weight, loss of, cyclic vomiting causing, 136
  - hypertrophic pyloric stenosis causing, 117, 124
- Whip-worm, intestinal infestation with, 304
- Widal reaction, significance of, 342
  - typhoid fever and, 191
- Worms, benign jaundice form, 328
  - intestinal infestation with, 296-309
- X-ray, gastro-intestinal diagnosis and, 335









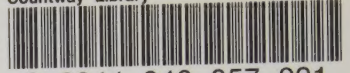
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